This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

WIDENER
HN UWEZ M

3.

Harvard College Library

BOUGHT WITH MONEY
RECEIVED FROM THE
SALE OF DUPLICATES

THE
Practical Medicine Series

COMPRISING TEN VOLUMES ON THE YEAR'S PROGRESS
IN MEDICINE AND SURGERY

UNDER THE GENERAL EDITORIAL CHARGE OF

GUSTAVUS P. HEAD, M. D.
PROFESSOR OF LARYNGOLOGY AND RHINOLOGY,
CHICAGO POST-GRADUATE MEDICAL SCHOOL

CHARLES L. MIX, A. M., M. D.
PROFESSOR OF PHYSICAL DIAGNOSIS IN THE
NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

VOLUME X

NERVOUS AND MENTAL DISEASES

EDITED BY

HUGH T. PATRICK, M. D.
PROFESSOR OF NEUROLOGY IN THE CHICAGO POLICLINIC. CLINICAL PROFESSOR
OF NERVOUS DISEASES IN THE NORTHWESTERN UNIVERSITY MEDICAL
SCHOOL; EX-PRESIDENT CHICAGO NEUROLOGICAL SOCIETY.

PETER BASSOE, M. D.
ASSISTANT PROFESSOR OF NERVOUS AND MENTAL DISEASES,
RUSH MEDICAL COLLEGE.

SERIES 1912

CHICAGO
THE YEAR BOOK PUBLISHERS
180 N. Dearborn Street

Phil 22.6.

HARVARD COLLEGE
APR 13 1918
LIBRARY

Duplicate money

COPYRIGHT 1913
BY
THE YEAR BOOK PUBLISHERS

HARVARD COLLEGE
LIBRARY

TABLE OF CONTENTS.

DISEASES OF THE NERVOUS SYSTEM.

Page
Symptomatology 5
The Neuroses 10
  Psycho-Analysis 10
  Epilepsy 20
  Other Neuroses 32

Diseases of the Brain and Meninges 38
  The Meninges 38
  Cerebral Localization 43
  Cerebral Spastic Paralysis 55
  Cerebral Syphilis 59
  Brain Tumor 74
  The Basal Ganglia 91
  The Hypophysis 101
  The Pineal Body 109

Diseases of the Spinal Cord 111
  Acute Anterior Poliomyelitis 111
  Tuberculosis of the Spinal Cord 134
  Tabes Dorsalis 136
  Lateral Sclerosis 137
  Multiple Sclerosis 138
  Traumatic Affections of the Cord 141
  Intermittent Claudication 145
  Spinal Cord Decompression 147

Diseases of the Peripheral Nerves 152
  The Cranial Nerves 152
  The Spinal Nerves 159
  Multiple Neuritis 165
  Neurofibromatosis 174
  The Sympathetic Nerves 176

Diseases of the Muscles 177

MISCELLANEOUS 182
Tobacco and the Nervous System 182
Hemihypertrophies and Hemiatrophies 187
Angioneurotic Edema 193
Paralysis Agitans 195
Sunstroke 198
Electrocution 202
Pellagra 202

MENTAL DISEASES.

General Considerations 205
Dementia Precox 214
Paretic Dementia 216
Other Psychoses 220

DISEASES OF THE NERVOUS SYSTEM.

SYMPTOMATOLOGY.

A New Reflex: the Wrist Sign. In the *Practical Medicine Series,* 1911, Vol. X, p. 7, a new reflex called by its discoverer the external malleolar sign, was described. This investigator, Chaddock,[1] of St. Louis, now describes another new reflex, in the upper extremity, which in a way is analogous to the external malleolar. "An area on the wrist which seemed to me to correspond anatomically with the external malleolar skin-area, I thought a promising field for investigation. The place I chose is located on the palmar surface of the wrist at the junction of the palm with the wrist, and just at the ulnar side of the tendon of the *flexor carpi radialis* and the tendon of the *palmaris longus,* evident as a groove or depression when the hand and fingers are in moderate flexion. My experiments have seemed to show that from the skin of this groove, as far distally as the base of the thenar eminence in the palm, and proximally an inch or so up the bend of the wrist, a peculiar reflex movement can be excited in case of disease of the corresponding pyramidal tract at a level higher than related spinal centers in the cervical enlargement of the cord. Indeed, in a few instances, irritation of almost any area of the skin of the forearm, as high as the elbow on the ulnar side, has induced the movement.

"The form of irritation I have used is scratching with a dull-pointed orangewood stick, or with a dull-pointed nail-file as shown in the photograph. [Plate I.] In a few cases, pressure with the point of the instrument, without scratching, has proved effective.

"The movement I have observed consists of flexion of the wrist and simultaneous extension and separation

---

(1) Interstate Med. Jour., February, 1912.

of all the digits. In certain cases this movement occurs slowly, and the resulting attitude is maintained for a few moments while the stimulus is continued; in other cases, it occurs very quickly and sharply, even occasionally with observable nervous tremor. In addition to this movement there is frequently, if not invariably, a simultaneous contraction of the triceps muscle, limited to a few of its middle fibers as a rule, but in some cases amounting to a decided bulging of the whole muscle. It will be noted that there is here a certain correspondence with the movement of thigh muscles excitable from the sole and external malleolar skin-area; and also that the induction of this movement, in some cases, from a wide area of the skin of the forearm, is analogous to abnormal toe-signs excitable from extensive skin-areas of the lower extremities.''

Chaddock has not found this reflex present in normal persons but has observed it in many persons in whom there was evidence of disease of the motor tracts, such as paretic dementia, original epilepsy, organic hemiplegia, multiple sclerosis, tuberculous meningitis, cerebrospinal syphilis, Sydenham's chorea and traumatic cerebral hemorrhage. Chaddock has not found any relation to exist between this sign and the finger-phenomenon of Gordon.

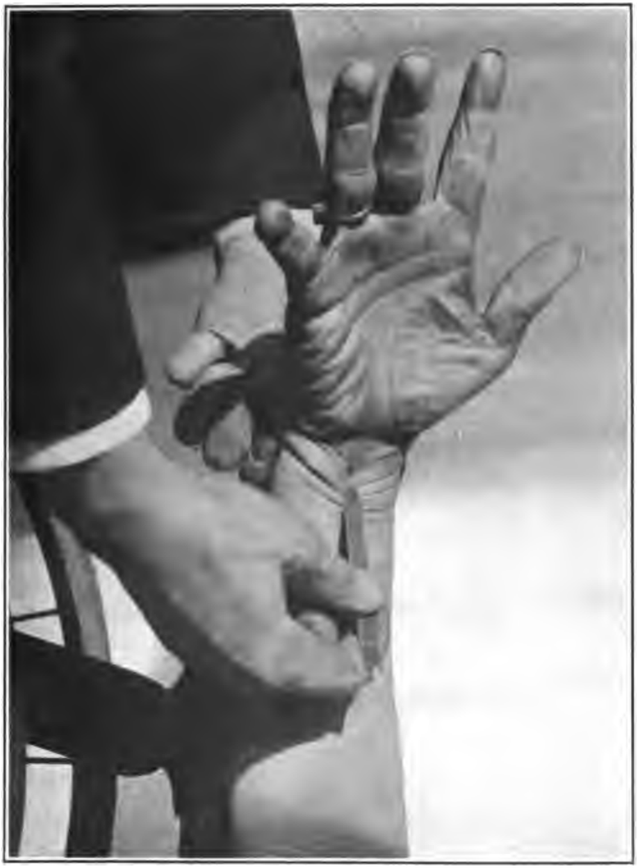
The Cytology of the Spinal Fluid. While the value and significance of the presence of various types of leukocytes in the spinal fluid are universally known, the finding of tumor cells in recognizable form is comparatively unusual. This subject is discussed by Richard Mohr[2] who also reports a case in which, for the first time to his knowledge, cholesterin crystals were demonstrated.

The patient was a man 46 years old. Five years ago he began to have pain in the lower portion of the spine and later in the left lower extremity. He was first treated for sciatica, but after a year the right leg became involved, as well as the genital and anal regions. Two years ago bladder disturbance set in, and the weakness of the legs gradually progressed to complete paralysis. Syphilis was denied and the Wasser-

---

(2) Deutsche Zeitschr. f. Nervenheilk., Vol. 44, p. 418.

PLATE I.



Method of eliciting wrist sign.—Chaddock. (Page 5.)

mann test was negative. The knee- and ankle-jerks as well as the plantar and anal reflexes were absent. Sensation was lost in the feet, posterior portions of the legs and thighs, buttocks, scrotum, and outside of the legs. In other words there was anesthesia below the level of the fifth lumbar segment. Naturally these findings led to a diagnosis of tumor of the cauda equina. Lumbar puncture yielded a clear, yellowish fluid containing cholesterin crystals and numerous large rounded cells filled with fat globules. The nuclei were very indistinct, and the cells evidently were much degenerated. The cells were too large to be fat granule cells derived from the cord, and were undoubtedly tumor cells. The cholesterin is considered to be derived from the liberated fat in cells which already had been destroyed. There was no increase in albumin.

In his interesting review of the literature the author points out that Krönig found free myelin, pieces of medullated fibers and hematoidin crystals in the spinal fluid of four patients in whom necropsies later revealed the existence of foci of softening in the brain, which foci had broken through the pia into the subarachnoid space. Rindfleisch was probably the first to find tumor cells in the spinal fluid in a case of diffuse sarcoma of the pia. The cells were large and almost filled by the nucleus. The fluid was rich in albumin, of yellowish color, and coagulated spontaneously. The necropsy showed that the tumor consisted of cells like those found in the spinal fluid.

Other authors also have reported cases of sarcoma and endothelioma with yellow fluids containing tumor cells. Schwarz and Bartels[3] found epithelial-like cells in a case of diffuse carcinoma of the meninges. Stadelmann found carcinomatous cells in the spinal fluid of a case of metastatic carcinoma from a primary growth in the stomach, and E. Meyer found tumor cells in the clear, uncolored spinal fluid taken from a case of sarcoma of the falx cerebri.

Diacetic Acid in the Cerebrospinal Fluid was first demonstrated by Grunberger[4] in a case of diabetic

---

(3) Practical Medicine Series, 1911, Vol. 10, p. 67.
(4) Centralbl. f. innere Med., 1905, p. 617.

coma. Later, Reichmann[5] found both acetone and diacetic acid in diabetic coma. Recently Richard Mohr also found acetone and diacetic acid in a diabetic patient, without coma, but in whose urine these substances were present in large amount. This finding, according to the author, is not surprising, as the very diffusible acetone bodies also are commonly found in the blood-serum and in the expired air, and occasionally in the contents of the stomach and intestine. The author does not attribute any special significance to the appearance of acetone in the spinal fluid.

Increase of Albumins and Xanthochromia of the Spinal Fluid, and their importance in the diagnosis of compression of the cord are discussed by Wilhelm Raven[6] who had opportunity to study 15 cases in which the diagnosis was verified by operation. His conclusions are:

1. Increase of albumin in the spinal fluid, together with little or no increase in cells speaks with great probability for compression of the cord.
2. A case of compression cannot be determined by the symptoms alone.
3. This syndrome may also be present in cases of intramedullary tumor.
4. The increase in albumin is caused by stasis and only below the place of compression.
5. Xanthochromia is due to blood pigment.
6. Xanthochromia is only of value in the diagnosis of compression of the cord, together with increased albumins, and then only in the absence of fresh hemorrhages and of meningomyelitic processes.

Chemical Tests of the Cerebrospinal Fluid. J. G. Greenfield[7] has studied the spinal fluid in 58 cases of nervous disease observed at the *Leeds General Infirmary.* He devised a quantitative method for the estimation of albumins by means of a modification of Noguchi's butyric acid test. The latter consists in boiling two parts of the fluid and five parts of 10-per-cent. butyric acid in salt solution. Then one part of normal sodium hydrate solution is added and the tube again boiled,

---

(5) Deutsche Zeitschr. f. Nervenheilk., Vol. 42, p. 1.
(6) Deutsche Zeitschr. f. Nervenheilk., Vol. 44, p. 380.
(7) Lancet, Sept. 7, 1912.

when a flocculent precipitate appears if the test is positive. In Greenfield's modification two c.c. of fluid are used, and after making the test the resulting mixture is poured into a graduated centrifuge tube which gives readings to 0.1 c.c. with fair accuracy. Each 0.1 c.c. by this method is equivalent to 0.025 per cent. He finds that normal fluids give an albumin-content of 0.05 to 0.2 c.c. (corresponding to 0.1 to 0.5 per 1,000), while cases of syphilitic meningitis gave readings up to 0.6 c.c. or 1.5 per 1,000.

Of special interest were certain cases showing high albumin-content, yellowish discoloration of the fluid and sometimes a tendency to spontaneous coagulation. French writers, notably Sicard, have previously described massive coagulation and xanthochromia of the fluid in syphilitic meningitis. Greenfield has two such syphilitic cases and also found the same condition of the fluid in a case of carcinoma of the meninges. A case of sarcoma of the meninges also showed greatly increased albumin-content. The author finds the albumin test of considerable practical value.

It is interesting to observe from a practical point of view that the finding of a high albumin-content in the cerebrospinal fluid (above 1.0 c.c. from 2 c.c. by the Noguchi test) seems to be in almost every case an indication for operative treatment. Where the cause is syphilitic meningitis much good may be done by promoting free circulation of the cerebrospinal fluid to the lumbar segments of the cord, and where a level of anesthesia or paraplegia is found the operation is frequently followed by very good results. In such cases a cyst of fluid is often found at the operation, pressing on the cord and practically acting as a tumor.

Apart from syphilitic cases, the diagnosis is limited to spinal tumor, or other compression paraplegia—*e. g.*, Pott's disease, fracture, dislocation, etc.,—where the clinical history would make the diagnosis simple. Pachymeningitis cervicalis hypertrophica might possibly cause a similar condition of the fluid, and in this condition, as shown by Horsley's results, operative treatment is often extremely beneficial.

Color Hearing. G. Marinesco [8] has written a monograph on this strange phenomenon and relates a large number of cases. One was a woman 35 years old with neuropathic heredity who remembers having had the condition since the age of 6 years. Sensations of colors were associated with consonants, vowels, names of people, cities, countries, days and months and ordinary abstract or concrete nouns. Certain letters and words appear to her written in color or on a colored background while others were associated with colored images. The letter *O* was associated with a small globe with the color of a cloud illuminated by the moonlight. The colors associated with abstract words were the most vivid. Music produced olfactory sensations in her.

Another patient was a young man of marked psychopathic heredity who had himself presented mental symptoms and was very musical, but insensitive to certain tastes such as that of quinine. With him also names and ciphers caused color sensations. His sister presented the phenomenon only in connection with the letters *B* and *G*. It has not been definitely decided whether this is a pathologic phenomenon or simply a psychologic peculiarity. The author believes that there is both auditory and visual hyperesthesia and diminished reciprocal inhibition in connection with the corresponding centers.

THE NEUROSES.

PSYCHO-ANALYSIS.

Freud's Psycho-Analytic Method and its Evolution is the title of an admirable address by the prominent Boston neurologist, James J. Putnam,[9] who begins by commenting on the high character and earnestness of the leaders of this new movement. On visiting them in Europe he learned that they had subjected themselves to a searching character analysis in preparation for their work, and for this reason: "The main thesis of the supporters of these new doctrines—which are at bottom old doctrines, re-arranged and re-emphasized, for psycho-analysis is largely an accentuated phase of education,—is that most of the emotional disorders to which we give the name of psychoneuroses arise largely from a more or less instinctive self-concealment, and concealment of one's self from others; that is, from an unwillingness or an inability to see or look at all the facts that should be seen, as the basis of the best feeling, thought and action. Recognizing this principle these physicians have seen that so long as their own lives, too, are partially on a false basis, so long as they also are self-concealed, they cannot do justice to their patients, either in the way of appreciation or of criticism.''

---

[8] Annal. de l'Acad. Roman., Vol. 33, 1911. Abstract in Revue Neurolog., Jan. 15, 1912.

[9] Boston Med. and Surg. Jour., Jan. 25, 1912.

Putnam describes psycho-analysis as a method of investigation and treating nervous invalidism and (incidentally) faults of character, which owes its strength to the fact that it searches and studies in detail, so far as this is practicable, all the significant experiences through which the patient to be treated has passed, and the motives and impulses which have animated him at psychologically important moments of his life, ever since his earliest childhood. In doing this it discovers, not, indeed, all the causes of the disorder from which he suffers, but a large number of important partial causes, and thus prepares the way for the influences tending toward recovery.

During the psycho-analytic treatment special effort is made to bring out those motives and impulses that were originally based on emotions which were repressed because they were painful or seemed out of harmony with the chosen plan of life, but which, in spite of all repression, had remained as active causes of serious mischief. The practical aim is to enable persons who are hampered by nervous symptoms and faults of character to make themselves more efficient members of society, by teaching them to shake themselves free from the subtle web of delusive, misleading, half-unconscious ideas and feelings by which they are bound and blinded as if through the influence of an evil spell. Such persons—and in some measure the statement is true of all persons—have to learn that they are responsible, not only for the visible, but also for the hidden portions of themselves, and that, hard as the task may be, they should learn to know themselves thoroughly in this sense. For it is the whole of ourselves that acts, and we are responsible for the supervision of the unseen as well as for the obvious factors that are at work. The moon may be only half illuminated and half visible, but the invisible half goes on, none the less, exerting its share of influence on the motion of the tides and earth.

After a brief discussion of the word-association method of Jung he takes up the most important part of the procedure, namely the analysis of dreams. He points out how our dreams may be utilized to the important end of bridging over the undesirable disharmony between the conscious and unconscious portions of our lives. These hidden portions of our lives must be thought of as seeking to make themselves felt, in action if not in words. Ordinarily, we keep them, like the evil spirits in Pandora's box, under pretty strong lock and key. At night, however, the locks are loosened, and our repressed emotions succeed in finding their way to the theater-stage of the dreamer's consciousness. Even then, the thoughts which arise are not allowed to become too evident, but are concealed beneath picturesque symbolisms and disguises. It is a very interesting fact that, as each new person comes into the world and begins his life of dreams, he adopts forms of symbolism analogous to those which have been in use since even semi-civilized life began. The various animals with which our childhood was familiar come forward to play the rôle of animal-passions; the rapidly moving trains typify our hurrying emotions. And so, too, water, the rooms or buildings in which we like to place ourselves, the bare or varied landscapes and many a symbol more, are all utilized as elements of a picture-language which is almost as well defined as that indicated by the rebuses of the child, or the hieroglyphs of the Egyptians, or in the mythology of the ancient Greeks. So full of meaning are these signs that no dream carries its true, much less its whole, significance on its face; no item, no obvious omission even, is without its bearing; no feature or character is to be found that is not of multiple value.

The general proposition has been laid down—and certainly with good reason—that every dream represents the fulfillment of an unconscious wish, of course in the same sense that this is true (as it unquestionably is) of the day-dreams of childhood. In the dreams which appear so terrifying, the wish is concealed behind an attempt to repress it, just as the partial wishes of our waking moments are often concealed behind the disguise of fears, a phenomenon very characteristic of the phobias of neurotic patients.

This belief in the rôle of dreams as important links in our innermost mental life is one of the essential pillars of the Freudian structure, and it is therefore eminently proper for Putnam to bring forth some arguments against the common notion that dreams merely consist of a jumble of trivial happenings of the day before. It is true that every dream takes the happenings of the day before as materials out of which to construct its story. These trivial experiences are utilizable, partly because of their analogic bearings, partly because they are still conveniently available by the memory and yet not fully woven into any other of the various complexes of which our mental furniture is composed. In utilizing these experiences the dreamer does what any person might do who wished to tell a story while sitting at the dinner-table with his friends. Assuming that he desired to describe a journey he had taken, he might select a salt-cellar to stand for a castle that he had seen, a fork for one road, a spoon for another road, a plate for a pond or lake, etc. But behind these hastily chosen symbols, there would be a connected story; and in the same way, behind the trivial details which make the outward framework of the dream, there is a connected story, which, indeed, reaches, in layer after layer, back into the dreamer's earlier life and even into his childhood. For in every mental act the whole personality of the individual comes into play, although in each act certain elements of the personality are illustrated far more than all the rest. Of course, it need hardly be said that the analogy between the forks and spoons and the apparently trivial incidents of the day previous to the dream-night is by no means a complete one. Unimportant as the real incidents may seem they are often full of meaning, which, however, only an expert analysis can reveal. Each dream, then, furnishes, to the expert, and to the patient, a path into the inmost recesses of the patient's life, better than any other means could furnish.

Putnam points out that the method of psycho-analysis has its special limitations. The patient, to be treated with success, must be reasonably young, reasonably intelligent and able to give a large amount of continuous time to the investigation. His outlook as regards conditions of life must be reasonably favorable, or else he must have the capacity for idealization such as will enable him to override outward misfortunes, and to face conditions cheerfully as they are. He must want to get well, and not count on his illness as giving him gratifications or advantages which he is unwilling to sacrifice, even for better health. Then, of course, some sorts of symptoms are less curable than others.

The length of time sometimes required for successful treatments has often been the subject of comment. But in fact it is a great gain to have a method capable, even in a long time, of producing fairly good results. Any one who thinks about the matter must realize that it is extremely difficult to make any considerable change in one's own character or habits. Our good qualities, as well as our faults, are deeply founded. Both have their roots in the experiences of infancy or in the reactions of childhood, and if we would help ourselves to the best purpose we must get back, in knowledge, feeling and imagination, to the conditions under which the deviations from the normal first began. To accomplish this takes time and patience, though the task is full of interest.

The Application of Psycho-Analysis to Insanity is recommended by C. Macfie Campbell.[1] The following case with comments by the author shows how certain apparently vulgar symptoms may have a deep meaning: A young man, 26 years of age, came under obser-

---

(1) N. Y. Med. Jour., May 25, 1912.

vation, after he had for a period of over four years presented the symptoms of a common form of insanity. His mood alternated between depression and elation. At one period he carried a revolver to defend himself; he said that he had been drugged and operated on in a hypnotic state, persecuted by Jesuits and Tammany men. He heard imaginary voices and tried to commit suicide.

Several of the delusions of this patient referred to bodily complaints. He thought that he could not chew; he attributed all his trouble to constipation; he wished to be allowed to fast for forty days. He was much troubled by a feeling of heat in the head, a drawing in his brain. The analysis of the patient's utterances on the subject of constipation, and of the value he laid on fasting, led directly down into *the depths of his inner life* and back to *the early stages of his development;* these two expressions are intimately related because the depths of consciousness are only intelligible in relation to early childhood feelings, fancies, beliefs and experiences. The patient himself brought his constipation into direct relation with childhood trends. He said that he had been a moody child and referred to "a peculiar feeling of uncertainty, doubt, and dread —a feeling of horror which was centered around the anus and the ileo-cecal valve—it felt as if the tube and the convolutions of the intestines were vibrating, from the throat to the intestines—I was later told that this was chronic constipation," an excellent example of how gladly the personality welcomes a current term, a familiar diagnosis, to veil the significance of uncongenial trends. It is evident in this case that we are not dealing with an ordinary insufficiency of the colon or the rectum, and the meaning of the so-called constipation is clearer when we find the patient using the same terms to describe his sensations when embarrassed in the presence of a buxom teacher. The patient clamored for cathartics and insisted on the great significance of his long fasts, but the real disorder from which he vaguely longed to purge his system was not one to be reached by drugs. Although distressed by the claims of the sexual instinct, he posed as an ascetic and freely expressed his contempt for woman, yet he evidently dwelt on sexual topics with keen pleasure.

The following statement of the patient illustrates the confused symbolical level of thought, at which various trends are brought together in strange juxtaposition: "Cleanliness is next to Godliness. Guts come from the German word *Götter,* the word for God."

The patient had a habit of running water on his head for fifteen minues at a time, and this was explained to be due to a feeling of heat in his head. He hated women, because by one of their sex he was early stimulated and made to have the heated feeling in his head. This feeling interfered with study; also, he could not study with his sister in the room, and the root of this lay in childhood experiences in which his sister had played a rôle.

The patient laid stress on a feeling of dread and horror once associated with a serpentine movement at the anal aperture, when he passed what he described as a snake or a serpent, but also as a tapeworm.

The full significance of such utterances is appreciated only in the light of similar cases; thus a young woman, with a case of dementia precox, deliberately vomited for two weeks because there was a lizard in her stomach, but she also said there was a man in her stomach. Another woman had a peculiar cough and complained of something in her throat. She hesitated to cough in case she should hurt Christ. Christ was in her stomach, she had taken in Christ at Communion; but again she described the throat-feeling as that of a worm creeping down her throat; it was the kind of worm that every man had. No case could illustrate more strikingly the level of thought at which many of the symptoms of mental disorders receive their explanation.

Anal Eroticism is the disagreeable but remarkable subject of a very lucid analysis by A. A. Brill[2] who is one of the foremost exponents of Freud's ideas in America. Some years ago Freud pointed out that in infancy and early childhood many portions of the body furnish sensations which contribute to the dawning sexual feeling and constitute "erogenic zones." Aside

---

(2) Jour. Abn. Psych., August-September, 1912.

from the sexual organs proper and the bladder outlet the mouth and anus are such zones which later normally are deflected from sexual aims and utilized for other purposes. In certain individuals, however, peculiar reactions occur and a peculiar character develops, which features together constitute the "anal complex." Freud has described a "triad" in such people, consisting of orderliness, economy and obstinacy. They usually will tell that as children it took them a long time to learn to control their bowels and that it gave them a peculiar pleasure to withhold their passages as long as possible. This is supposed to give rise to economy to the point of avarice in later life, fondness for feces changing into fondness for gold. It has long been noted that insane patients associate feces with gold and the term "filthy lucre" is thought to indicate such association in the popular mind. Three very remarkable cases are reported by Brill.

Case 1. A successful merchant, 44 years old, had suffered from distressing compulsive ideas since the age of 20. When eating soup he would think that it was urine and sausages always reminded him of feces. The noise of an automobile horn made him think of flatus to such an intolerable degree that he gave up automobile riding. A woman's mouth made him think of the rectum and her eyes recalled the anus. Shaking hands with a man made him think of a person using toilet paper. A person with prominent teeth to him always recalled feces protruding from the anus. Although wealthy he was a typical miser and extremely small in all money matters. During psycho-analysis it was learned that as a child the patient had had a hard time to control his rectum, and that he had been punished and jeered at for soiling himself up to his sixth year. At nine years he was sent home from school in disgrace for having audibly expelled flatus in the class-room. He always took great interest in his anal region and in the feces. When he first came to Brill he promised him 75 per cent. of his income if he could be cured of the obsession caused by automobile horns, and after a few weeks of psycho-analysis, this, and some other obsessions were actually removed but his avarice got the best of him and he did not pay even a moderate fee.

Case 2. A man of 30 years suffered from obsessions, doubts and fears. He could not resist the impulse to rub his feces on walls and at times on his body. He formed the habit of holding back his feces, which gave him pleasure and stimulated his mental activity, so when given a difficult task he "practiced constipation." Thus as a reporter he was once sent to describe the maneuvers of the National Guard and in order to do it really well he held his bowel movements back for two or three days until it became almost unbearable, and he would then imagine himself on the battlefield of Waterloo and would describe what he saw.

Case 3. A man of 39 years was obsessed with doubts and phobias which referred to definite ideas about people being killed. He had showed an enhanced anal activity in childhood, but the most prominent factor in his infantile sexuality was the component of cruelty. His greatest pleasure up to the age of ten years was shooting birds, rabbits and squirrels. At the age of puberty he became very sympathetic, and one day after shooting a squirrel he suddenly experienced feelings of compassion and remorse. Since then he found it hard to go out shooting. When his neurosis developed at the age of 18 years he began to be troubled with constipation of obstinate character which resisted medication and persisted for 15 years until he accidentally discovered the following remarkable psycho-therapeutic process.

He was playing with a spool of cotton upon which there was a picture of a child. He rolled it and when the child's picture faced him he stuck a pin into it. Five minutes later he had a bowel movement and from that time on he could produce a movement by going through this process which he gradually modified. He carried a number of long pins which he sharpened from time to time, and every morning he drew a picture of a girl and thrust the pins into the region of the heart. When he was very busy he would simply draw a target on paper and throw a pin at it, imagining that it was a girl. As the years went by he resorted to many variations. Thus when he lived in the country he would shoulder his rifle and go out into the garden, and by imagining that he was shooting Indians his bowels were soon stimulated to activity. Sometimes he imagined himself fighting which gave the same result. On one occasion while throwing his pins at a picture one of them fell through the window into the garden, and as children were wont to play there he soon became obsessed by the idea that one of the children might swallow the pin and die. This was the first obsession of this kind which continued in different forms.

Brill concludes his paper with the following general remarks: "All these patients show a special interest in their anal activities in childhood and in adult life. Later when the infantile activities of the anal zone remained in a state of repression they belonged to that class of persons who prolong the act of defecation by reading books and newspapers in the water closet; thus case 1 referred to the water-closet as his library. With the onset of the neurosis, which signifies a failure of repression, the originally enhanced anal activities came to the surface in the form of symptoms; *i. e.*, the neurosis represented the negative of the perversion. The analysis of these as well as of a number of other cases fully corroborate Freud's formula; *viz.*, that the permanent distinguishing traits of a person are either unchanged continuations of the original impulses, sublimations of the same, or reactions formed against them."

The Value of Psycho-Analysis in the Correction of Defects of Character is found to be great by E. W. Scripture (3) who says:

"Among the methods of therapy for character defects, psycho-analysis is certainly one of the most effective. I have had boys and young men living with me in the closest intimacy for months and years without ever being able to know them as thoroughly as now after a few weeks of psycho-analysis. There are two reasons for this: psycho-analysis brings about an intimacy that cannot be attained in any other way and it reveals the traits of character that would otherwise remain unknown to anyone. The analysis itself is a vital part of the cure; as the patient recognizes his abnormalities in this way he instinctively corrects them. It is most effective to combine psycho-analysis with educative treatment.

---

(3) Med. Record, Oct. 28, 1911.

Various objections have been raised to psycho-analysis. The most frequent is that there is too much talk about sexual matters. Such discussion is, however, often entirely unnecessary. When it occurs, it can be done with such delicacy as not to offend the most modest person.

Another criticism of psycho-analysis is that it takes a great deal of time. This shows an entire misunderstanding of the case. Psycho-analysis is valuable for just the reason that in some cases it achieves results in a far shorter time than they can be attained in any other way, and in other cases it is successful where other methods are hopeless. In the treatment of character improvement begins immediately and results are apparent from the start.

EPILEPSY.

Hygienic Versus Bromid Treatment. Under the title: "The Curability of Idiopathic Epilepsy with Report of 29 Cures" L. Pierce Clark[4] defines the criteria which should govern us in pronouncing a case cured, and gives an outline of the hygienic treatment which he advocates and which is in use at the *Craig Colony for Epileptics* where most of his cases were observed. He first points out that confusion exists as to what constitutes idiopathic epilepsy and still more as to what constitutes a cure. Regarding the first question, he states that the occurrence of repeated fits over a period of two years where no adequate organic lesion was present ought ordinarily to suffice to make a diagnosis of idiopathic epilepsy certain.

As to the second question, he says: "Inasmuch as the time limit constituting a cure in idiopathic epilepsy varies from 2 to 20 or even 30 years one needs to formulate some more definite data than a mere time criterion on which one may depend. Indeed it would be far better to eliminate the word 'cure' in this con-

---

(4) Archiv. Int. Med., January, 1912.

nection and substitute the term 'arrest.' Furthermore, the time element alone is as worthless as percentages of cures unaccompanied by actual case histories. One cannot arrive at anything definite regarding the permanency of cure or arrest without knowledge of the psychic and somatic factors of individual cases. For instance, not a few writers report cures when the patient has passed but a year or so of freedom from attacks although the patient still remains under sedative treatment. The cured patient must be able to assume charge of his or her affairs, assume the responsibility of life. He should have a true insight into the nature and conditions which brought about the disease. He should be able to direct his efforts to ward off the irregularities of life in the future. In short, a changed mental attitude toward himself and his environment marks the stability of an arrest far more than a mere cessation of fits. If the one-time patient does not become tractable and self-regulative, Clark does not hold that the cure will be permanent. The irritability must disappear, ambition and proper initiative must return, and above all the memory must show decided mending. In a physical way the digestion and processes of elimination must approximate the normal, even under slight digressions of diet.

As to curability Clark says: "Aside from the cases reported in this paper for the first time, there are but few authentic data at hand relative to cured idiopathic epilepsy. There is plenty of evidence and not a few statements at hand, showing that from 5 to 10 per cent. of idiopathic epileptics are cured, but careful case reports backing these figures do not exist. All of us should look to this defect. Aside from backing the statistical percentages of cures, careful case reports may teach us a more definite and scientific therapy of the disease."

As to curability at different ages, it is pointed out that epilepsy can scarcely be considered established in the so-called spontaneous cures which are reported to have taken place at the age of 4 or 5 years, because convulsions at this early age are so frequently of a reflex character. It is, however, significant that in nearly all such cases the hygienic plan of treatment has been used. Spontaneous cures rarely if ever occur between 5 or 6 years of age and the age of 20 years, a period commonly designated both by lay and professional advice as that in which a patient may be expected to outgrow his disease. The chances of spontaneous cure improve greatly with advancing years up to 45 years, after which few such cures are noted. Of his own 29 cases the age of arrest of attacks ranges from 2 to 17 years. The age of the youngest patient at the time of cessation of attacks was 8 years and that of the oldest was 48 years. The majority were in early adult life. The duration of treatment was from 3 to 8 years.

The age at the outset of the attacks varied from earliest infancy to 26 years. Fully two-thirds of the patients contracted the disease before the age of 15 years, and in two-thirds of the cases there was a family history of either epilepsy, or insanity, or both.

Clark's plan of treatment as outlined for a typical case in a young man of 20 is as follows: The patient is placed first of all with a nurse trainer in a congenial but quiet country environment, away from the family by preference. The daily schedule runs about as follows: The patient arises at 6 a. m. and takes 10 or 15 minutes of a gradually increasing severe grade of active calisthenics with Indian clubs, medicine ball or weights. He then takes a glass of hot water, a cold shower-bath and a brisk rub-down and breakfasts on an egg, a small amount of cooked cereal, a glass of milk and some fruit. Immediately after breakfast he takes a walk of six to eight miles which he covers in running and walking in about one and a half hours. He then takes a glass of milk with or without raw eggs and one or two strips of zwieback. He is then given a shower bath and lies down for 15 minutes and afterwards works at sawing wood or some equally useful occupation until 12:30 p. m. He then lies down without undressing, sleeps or not half an hour, and is then given a dinner of fish and milk, vegetables, cereal and stewed fruit. This meal should be eaten in not less than 40 minutes. He is then allowed to sleep for an hour after which he does woodsawing again as in the morning, and midafternoon lunch follows with rest. From 5 to 6 p. m. he is allowed to follow some light intellectual occupation, such as reading or study.

Supper of milk, eggs, cereal and stewed fruit is given at 6:30 p. m. This meal, like breakfast, is light and should occupy at least 30 minutes. The patient is then allowed games, recreation, etc., until 8:30 p. m. He then takes a warm bath and general massage, etc., and is in bed for the night at 9:30 p. m. No alcohol, tobacco, tea or coffee is allowed. The patient is encouraged to drink at least two quarts of water daily. High colon irrigations are given every other night until the colitis is reduced, and every ten days castor oil or calomel in divided doses is given. The articles of food in the three-meal-diet are changed every fortnight. The occupation is also changed every two weeks if desired. The patient is steadily encouraged to work and play at concert pitch: rest, complete and absolute, is also insisted on. No slack or taking it easy in the day's program is allowed.

The author contends that the introduction of sedatives in epilepsy has been fraught with more harm than good and that the sedatives have clouded the issues of causation and prognosis to a remarkable extent. Bromids mask the progress of the disease and make a full understanding of the forces at work in the individual case unbelievably complex. Clark has seen many patients treated by sedatives steadily advance in physical, mental and moral deterioration in the face of a steady cessation of fits. In not a few the account of a prolonged suppression of fits is squared by an outburst of status epilepticus or a furious maniacal attack. He sees only two uses for the bromids: (1) to determine the severity of the disease by noting the amount necessary to hold the fits in check and (2) as an adjuvant after all discoverable causes are set right, and in cases needing some small degree of sedation to bridge over periods of activity.

The Crotalin Treatment, or treatment with rattlesnake venom has been thoroughly tried out by Ralph H. Spangler,(5) who tabulates the results in 36 cases,

---

(5) N. Y. Med. Jour., Sept. 9, 1911.

The age of the patients ranged from 5 to 62 years. The solution used consists of the dried, crystal-like scales from the venom dissolved in sterile water, to which has been added a few drops of tricresol for its preservative and antiseptic effect. The solution was put into sterilized ampoules each containing one dose, and solutions of various strengths were made, namely 1/200, 1/100 and 1/75 of a grain of venom. The strength of the dose must be determined for each individual so it is best to start with 1/200 of a grain and note the local reaction which is very variable. The author gives the injections in the forearm. The injections are given a few days apart so as to give the local reaction time to disappear. In the majority of cases all other medication was withdrawn. The results according to the table compare very favorably with other forms of treatment. Four of the thirty-six patients have had no attacks since the treatment was given, although they have been observed as long as two years. In most cases the attacks were lessened in number and severity and the mental and physical condition of the patients improved. As to diet, the patients were allowed a general mixed diet but they were warned to avoid such articles of food as were found to produce gastro-intestinal disturbance. For laxatives, cascara sagrada or mineral waters were used.

In a later article[6] the same author further discusses the treatment which he now has used in 109 cases with over 2,000 injections. Further details are given of 6 of the cases previously reported, several of which have remained free from attacks. Eight new cases are related with some detail and the chemistry of the venom and its action on the coagulability of the blood are discussed. The coagulability is decreased, and in this connection it is of interest that John Turner[7] arrived at the following conclusions from his work on the coagulability of the blood in epilepsy: (a) The average rate of coagulation of the blood is greater in severe cases of epilepsy; (b) the rate is quickened during the period that the patient is having fits (serial epilepsy); (c) there is a further quickening of the coagulation rate up to twenty-

---

(6) N. Y. Med. Jour., Sept. 14, 1912.
(7) Jour. Mental Science, October, 1908.

four hours before a seizure; (d) from twenty-four to forty-eight hours after a fit, there is a rebound and retardation in the rate of blood coagulation.

Spangler's personal investigation in 18 cases so far have largely confirmed Turner's conclusions. Moreover the effect of the venom injections if given over a period of time seems to be permanently to decrease the rate of coagulability of the blood. Further work on this subject is in progress and will be published later. The author's presentation of the crotalin treatment is summarized by him as follows:

1. The "venom treatment" is indicated in many of the essential cases of epilepsy.
2. Not only are the virulence and number of epileptic fits favorably influenced by the crotalin treatment, but the excitability of the nervous system is modified and the general health of the patients, their mental faculties, and metabolism in every respect are considerably improved.
3. The quality of the blood, and possibly its chemical composition seem to be affected by the injection of the venom. As to the exact effect it has on the coagulability of the blood, further observation is necessary.
4. There is no danger in the use of crotalin as long as the necessary aseptic precautions are taken in its administration, and the treatment is carried out with careful observation of its effect on the patient.

Surgical Treatment. Charles H. Frazier[8] makes a valuable contribution based on 63 personal cases. He first calls attention to the present unsettled condition as regards surgical indications. It is commonly stated that there is no demonstrable lesion of the cortex, either macroscopic or microscopic, in so-called essential epilepsy and that in cases of focal or Jacksonian epilepsy there is frequently a local lesion, but there are a great many exceptions to these rules. In his own cases the findings were entirely negative in only 6, and the lesions found were very diverse, such as cysts, adhesions, pachymeningitis with adhesions, edema or cysts, edema alone, edema with cyst, thickened dura, organized blood-clot, cystic degeneration and cortical gliosis. About one-half

---

(8) Therap. Gaz., March, 1912.

of the cases were classified as pachymeningitis or edema. He makes the following summary of his conclusions:

1. The development of the surgical treatment of epilepsy has met with many impediments, among which should be mentioned: (a) The uncertainty as to the origin of the attacks; (b) the multiplicity of the pathologic lesions; (c) the positive findings in some cases and the negative findings in others.

2. In a larger percentage of cases than has been hitherto appreciated, some pathologic lesion of the exposed cortex has been found.

3. Operative intervention should be regarded as justifiable: (a) In traumatic epilepsy with external evidence of an injury; (b) in traumatic epilepsy without external evidence of an injury when the nature of the attacks or the symptoms immediately following the injury indicate the seat of the lesion; (c) in all forms of Jacksonian epilepsy of whatever origin; (d) in general epilepsy where the suggestion of a focal lesion may be found by a careful physical examination before or after the attacks in some disturbance of motion, sensation or reflexes.

4. The operative procedure must be adapted to the character of the lesion: (a) Cranial defects should be repaired; (b) focal lesions, such as cysts, tumors, etc., should be removed; (c) edema, a very common lesion, calls for some method of drainage; (d) in Jacksonian cases without lesion the cortical center should be excised; (e) in idiopathic epilepsy without focal symptoms or lesions Kocher's decompression deserves consideration.

5. The value of the operation may be measured in terms of improvement or cure. Either should be considered as justifying the operation.

6. Gratifying results may be anticipated in at least 10 to 25 per cent. of cases.

In judging the results Frazier states that no case should be reported as cured unless at least three years have elapsed since the operation. Of 25 cases which have passed the three-year limit and which could be traced, 7, or 28 per cent., have been profoundly influenced by the operation. The first patient was a case of Jacksonian epilepsy of 5 months' duration in which a subcortical cyst was found at the operation and the cyst wall removed. He recovered considerable strength in the arm and leg which were partially paralyzed; the headaches of which he complained were entirely relieved and his attacks were reduced in frequency to one every two months and are much less severe than they were before.

In the second case the lesions were pachymeningitis, edema and increased tension. The operation was performed three years ago. There have been no attacks for the past three years and the patient was greatly improved in mental vigor.

The third patient was a young man 20 years old. Since the operation in 1906 the attacks have become progressively less severe and less frequent. He had been entirely free from attacks for the last nine months before the time of the report. Diffuse pachymeningitis and a cortical cyst were found at the operation. The patient's disposition has improved, he is able to work and in every other respect is perfectly well.

The fourth patient within a year of the operation in 1907 had sustained a serious cerebral contusion without focal symptoms, but with loss of consciousness followed by delirium extending over a period of several weeks, and an organized blood-clot was found in the motor region and removed. At the site of the clot there were firm adhesions between the dura and pia. Four years have elapsed since the operation, and in this time there have been no attacks for the past three and a half years. This case emphasizes the importance of operating upon all cases of cranial trauma in which there is any reason to believe there has been a cortical hemorrhage. Had it been possible to detect any focal symptoms at the time of the injury Frazier would have urged operation without further delay, but despite frequent and careful examination no evidence of cortical hemorrhage was apparent.

In the fifth case of the series nothing was found at the operation but an intense edema; although the patient had sustained a fracture of the skull there was no evidence of any injury to the internal table or to the underlying meninges and cortex. Before the operation he was having two or three attacks a week, all general in character, but he has had none since.

In the sixth case the patient was 23 years old at the time of the operation, three years and nine months ago. She had had concussion of the brain at 11 years, her first convulsion at 15 years; the attacks were general in character. An area of adhesions 2 cm. in diameter was found a little behind the postcentral convolution. Although almost four years have elapsed, the patient has had but one attack, of which she had no intimation, as it was not severe and occurred at night.

In the seventh case the patient, a young man of 20, had sustained a fracture of the skull at the age of six. The operation, performed almost four years ago, revealed an intense edema and a pachymeningitis, and since that time he has had no convulsive seizures. He has been in splendid health and able to attend his work regularly during the four years intervening.

Briefly, then, of 25 cases operated upon more than three years ago, 7 have been profoundly influenced. The following statistical summary gives all the necessary details as to etiology, character of operation and findings:

1. Number of Cases	63
2. Number of Operations	53
3. Character of Operations:	
Craniotomy	21
Decompression	3
Craniotomy and decompression	20
König-Müller repair of defect	4
Evacuation of cyst	3
Clot removed	1
Excision of facial center	1
4. Sex:	
Males	41
Females	12
5. Ages According to Decades:	
0-10	7
10-20	16
20-30	18
30-40	10
40-50	2

6. Etiology:

Fracture	9
Cranial trauma	18
Intracranial hemorrhage (newborn)	4
Epidural hemorrhage	1
Cerebral hemorrhage	1
Tumor	1
Chronic meningitis	1
Infectious fevers	8
Undetermined	10

7. Character of Attacks:

General	31
Jacksonian	18
Jacksonian and general	4

8. Duration of Disease:

Less than 6 months	10
6 months to one year	3
1 year to 2 years	8
3 years	7
4 years	2
5 years	5
6 years	4
7 years	1
8 years	4
9 years	1
10 years	1
11 years	1
15 years	1
20 years	1
Duration unknown	4

9. Findings at Operation:

Cyst	8
Adhesions	6
Adhesions and pachymeningitis	2
Edema	11
Edema and pachymeningitis	8
Dura thickened	2
Organized blood-clot	1
Cyst and pachymeningitis	1
Cyst and edema	1
Cystic degeneration	2
Softening	2

Pachymeningitis	2
Negative findings	6

Skepticism as to Surgical Treatment. The skepticism which is common to neurologists and surgeons is well expressed by C. H. Frazier[1] in another article:

"The problems that present themselves to the surgeon in the treatment of epilepsy are manifold, and the results, if the truth were known, are disheartening. At various times I have operated upon 53 epileptic subjects, presenting all the varied types of the disease including idiopathic epilepsy, Jacksonian and traumatic epilepsy, and petit mal. In the management of these cases it has been my custom to vary the procedure according to the individual case. In the Jacksonian type an exploratory craniotomy has been performed; in the absence of any lesion at all or any lesion amenable to surgical therapy, the flap was replaced, and in some instances the brain decompressed. In the traumatic type and in general epilepsy, if there was a history of trauma, no matter how remote the time of injury, the region of the trauma was explored, and again, in the absence of any demonstrable lesion, the brain decompressed. In a few cases of idiopathic epilepsy and petit mal cerebral decompression alone was practiced.

"My experience in this series of cases has shown that only in the most exceptional instances does one find a source of focal irritation, demonstrable to the naked eye, such, for example, as an organized blood-clot, adhesions, or a depressed fragment of bone. The mere finding of adhesions offers no solution to the problem, since there is no known means, either in the peritoneum or in the cerebral membrane, of preventing the readherence of surfaces once adherent. Likewise is it exceptional to find the brain of the epileptic subject perfectly normal in appearance. In the vast majority, no matter whether the attacks be general or focal, one sees at the operation very definite but diffuse pathologic lesions, such as edema or evidences of a pre-existing pachymeningitis, the two conditions very frequently associated. I allude to those phases of the pathologic

---

(1) Univ. Penn. Med. Bull., February, 1912.

anatomy of epilepsy merely to emphasize what is generally not appreciated: first, that in many instances there is a definite lesion; and, secondly, that this lesion is not one which can be favorably influenced by surgical therapy.

"It was at the suggestion of Kocher that cerebral decompression was first employed for epilepsy on the theory that immediately prior to the attacks there was a condition of hypertension. The establishment of an opening in the dura and skull, by preventing this condition, would serve as a prophylactic measure. Whether Kocher's hypothesis will ever prove to be founded on fact remains to be seen. I do know, however, that the operation he recommended, at least in my hands, has not furnished any evidence in its support, and, on the contrary, that attacks of epilepsy in those who have traumatic defects in the skull, following, *e. g.*, a comminuted fracture, have been relieved by repairing the defect.

"There are in my records 20 cases, in which, with or without an exploratory craniotomy, the brain was decompressed. Of these there is but one in which there have been no attacks since the operation; one had several attacks within a year of the operation, but none for several years; two are reported as being improved, but the remainder are either unimproved or the course of the disease has been in nowise influenced. These results are presented with the full realization that one sees, at times, more encouraging reports. But I am inclined to believe that in too many instances these so-called operative 'cures' are placed on record too soon, and that the results are merely those of operations *per se*, the freedom from attacks even for a year which may be observed when an epileptic subject has been operated upon, no matter how trivial, what the character, or where the seat of the operation.

"While the risk of operation is practically *nil*, my observations have led me to entertain a good deal of skepticism as to the future of the surgical treatment in this distressing condition. I have been forced to this conclusion partly by discouraging results and partly by observations made at the operations. It is my belief that epilepsy in most instances is not 'idiopathic,' in the sense that there is no demonstrable lesion, but, on the contrary, that in many there is a definite source of irritation for which there is as yet no surgical panacea."

"Surgeons with too exalted ideas as to the results of their operations upon epileptic subjects are referred respectfully to epileptic hospitals and colonies where I am reliably informed will be found not an inconsiderable number of their so-called operative cures."

OTHER NEUROSES.

Narcoleptic Attacks in Childhood Resembling Epilepsy are described by M. Friedmann.[2] As they offer a favorable prognosis, the differentiation from epilepsy is of great importance. Some years ago the author reported 4 cases in children between the ages of four and seven years. The attacks consisted of peculiar states of rigidity lasting ten to twenty seconds in which the eyes rolled upwards while the body was relaxed and could not be moved spontaneously, though automatic movements were possible and consciousness and memory were retained. The attacks were very frequent, from six to forty occurring every day, and they generally also occurred several times during the night, but when the patient was awake. Otherwise the children were normal and bright, and their nervous systems did not suffer in any other way. In 2 cases the affection lasted between seven and eight years. The author has seen 2 cases in adults who had suffered from similar attacks in childhood and he has also seen several cases beginning in adult life. He believes that the condition frequently occurs on a neurasthemie basis but that it has nothing to do with genuine epilepsy.

Recently he has seen 3 new cases. The first patient was a bright and lively girl 6 years old who had twenty to thirty attacks daily which seemed to have been brought on by excitement connected with her first term in school. She was greatly improved by two sojourns in the country and after two years the disease ceased. The second

---

(2) Neurolog. Centralbl., Nov. 1, 1911.

patient was a strong and bright girl with neuropathic heredity in whom the attacks came on after an operation on the nose. The attacks occurred at three different periods and each time ceased during a change of air. The third patient, a boy 8 years old, usually had the attacks when he first went out-doors in the morning and whenever he looked upward in the sunshine. Besides the upward rotation of the eyes there were a few nodding movements of the head. The attacks are not influenced by bromids and are generally brought on by some emotion. Medicinal treatment has so far been of no avail but a period of sojourn in the country, or rest in bed on account of some intercurrent disease, has produced cessation of the attacks. The affection is related to the narcolepsy described by Gelineau.

The Mental Symptoms of Chorea. P. Jolly[3] states that some degree of psychic disturbance must be said to exist in all cases of chorea minor. Of more severe mental disturbances delirious states are most frequent. He considers the choreic delirium an independent symptomatic group which most closely resembles acute amentia (confusional insanity) and acute paranoic conditions. As characteristic traits of the latter we have frequent changes in the delusions because they do not outlast the hallucinations with which they are connected, and there is a second stage of mild exaltation. Except in cases occurring in the second half of pregnancy, and in those associated with an unusually high degree of motor excitement and great loss of consciousness, the prognosis is good. Complete recovery without mental defect is the rule. Two cases are reported.

Case 1. A woman of 20 years with neuropathic heredity and who was naturally nervous developed right-sided choreic movements (hemichorea) after a mild attack of acute articular rheumatism. An old heart lesion was present. There was no fever. She was sometimes exalted and sometimes depressed and at times displayed fear. She had hallucinations of all the senses but was oriented as to time and place. After a month there was increasing inhibition, she stopped talking, had to be fed by a tube and had involuntary discharges. The

---

(3) Wien. klin. Wochenschr., No. 29, 1911.

hallucinations persisted but she remained oriented. After five months of this condition she passed through a state of exaltation which lasted for three months during which slight choreic movements still were present. She steadily gained in weight and finally made a complete recovery.

Case 2. A woman 27 years old, congenitally weakminded, whose father and sister had suffered from chorea following acute articular rheumatism developed chorea after a tooth extraction. After three weeks she became extremely excited, showed great fear, and had hallucinations. The choreic movements were extremely severe, she was unable to talk, had fever, rapid pulse, and slept very poorly. She died four days after admittance to the hospital. The necropsy showed no definite changes, the heart in particular being normal.

The Tendon Reflexes in Chorea Minor are discussed by L. E. Bregman.[4] In 1901 W. Gordon described a peculiarity of the knee-jerk observed by him in cases of chorea. The patient is placed in the recumbent position and while the muscles are relaxed as much as possible the knee-jerk is tested. It is then noted that the leg after being extended by the contraction of the quadriceps does not immediately drop down but remains raised for a few moments and then gradually sinks down to its previous position. This sign, while frequent in chorea, is not present in all cases and is not constantly present in a given case. It is most readily obtained in a mild case with only slight involuntary movements. It has received little attention in the literature. The author noticed it himself about two years ago before he knew that it had been described by others. He names it the "tonic reflex" and has found it present in nearly half of his cases of chorea. In hemichorea it occurs only on the affected side, and in cases where the movements are more marked on one side the "tonic reflex" is also more marked on that side. In some cases it does not occur until after the tendon has been tapped several times with the percussion hammer. The author has demonstrated a similar phenomenon in connection with other tendon reflexes in only one case.

---

(4) Neurolog. Centralbl., Nov. 16, 1911.

On testing the ankle-jerk the foot remains in plantar flexion for a time after the gastroenemius contraction has been produced by the tap on the Achilles tendon. Gordon looked upon this sign as produced by an involuntary choreic movement caused by the irritation of the testing of the reflex; that is, we are dealing with an "overflow of impulse" such as we also see in the voluntary movements of choreic patients, which are apt to be of unnecessary strength and amplitude or to give rise to superadded involuntary movements. Oppenheim gives a similar explanation. Bregman finds that the tonic contraction lasts longer than is usually the case with the choreic movements, and in one case observed for a long time it was noticed that choreic movements in the quadriceps were relatively rare and almost never caused a tonic extension of the leg as seen in connection with this sign. In the upper extremity where the choreic movements usually are much more pronounced he has never observed this sign. He believes it may be of value in differentiating between chorea and hysteria.

Unilateral Rigidity of the Pupil in Ophthalmic Migraine. Westphal[5] reports some very remarkable ocular phenomena in a nurse 21 years old who was in excellent general health. She began to menstruate at 17, but without apparent reason menstruation ceased from January, 1910 to December, 1911. At the time of cessation she developed attacks of severe left-sided headache with nausea, and the attacks were inaugurated by the appearance of scintillating scotomas. In May, 1911, Westphal observed an attack of left-sided headache in which the entire face was red and the left pupil was widely dilated and did not react either to light, in convergence, or consensually. At the same time the patient was completely aphonic and claimed to be almost unable to see with the left eye. Some days later, after the cessation of the headache, the pupil again behaved normally.

Within a week the patient was attacked by an insane woman and scratched in the face. She at once developed migraine attacks with marked redness of the left side of the head, and the left pupil again became widely

---

(5) Deutsche. med. Wochenschr., Sept. 19, 1912.

dilated and rigid. There was temporary left-sided blindness which was shown to be hysterical and was removed by suggestion. The left pupil remained wider for some days after the cessation of the headache. After a similar attack in August the left pupil remained dilated and rigid until December, and at times this pupil was not perfectly round. Almost every week she had attacks of left-sided headache and then claimed to see double. During the attacks the left cheek repeatedly was shown to be 6° to 7° warmer than the right one. On December 23 the left pupil had become narrower and reacted promptly to light, and on December 25 she again menstruated.

Very few such cases have been observed, while difference in the size of the pupils has been more frequently noted. Westphal finds only 3 cases in the literature similar to his own, and in all of these, dilatation of the pupil was attended by vasomotor disturbances, in two cases by hemorrhage in the orbit. The author believes that the paralysis of the sphincter of the iris was produced by vascular spasm or by pressure of hyperemic vessels on the pupillary fibers of the third nerve.

This pupillary condition is similar to one rarely occurring in katatonia and also described by Westphal. Thus one katatonic patient periodically showed rigidity and dilatation of the pupils for weeks at a time, while in the intervals the condition of the pupils was absolutely normal. The author believes that in the katatonic patient there may be in the muscular coats of the vessels a spastic condition analogous to that responsible for the rigid "kataleptic" state of the general musculature.

The Training-Camp Method in the Treatment of the Functional Neuroses is recommended, on the basis of personal experience with 30 cases, by Thomas J. Orbison[6] of Los Angeles. Among the cases related is that of a psychasthenic banker, 36 years old, who was troubled with many kinds of obsessions, a marked general feeling of inadequacy, lack of power of concentration, self-reproach, etc., so that he became unable to

---

(6) Jour. Amer. Med. Assoc., Jan. 13, 1912.

continue in his business. He was very hypochondriacal, suffered from gastro-intestinal atony, and his muscles were flabby. The treatment employed in this case and the result obtained were as follows:

He was sent at once to a training-camp in the foot-hills as he did not need a preliminary rest treatment. He received three months' rigid training. During this period the internal administration of such nerve tonics as valerian, hyoscyamus, camphor and asafetida, with cascara for the intestinal atony, was deemed advisable. At the end of that time he returned home and later resumed an active business life. The latest reports from him were very satisfactory.

In this case the method was applied as follows: The site of this training-camp was a cottage in the foot-hills near Pasadena. Here the patient and his trainer lived practically an out-door life. The trainer was a professional trainer of pugilists and athletic teams. The food was simple, nutritious and well-cooked. There was an open space at the rear of the cottage where boxing, wrestling, and medicine ball could be had regularly every afternoon. At 9:30 a. m. his trainer took him out for an hour of road work. The work was easy at first and gradually increased. After both work periods he was given a hardening rub-down (after a shower) and then was put to bed for a rest period of an hour. His whole day was arranged for him and all that was expected of him was to obey instructions.

The author's conclusions concerning this treatment are: "I have found this method to be a specific in the true neurasthenias — combined with other forms of rational therapeutics. Final results have been more gratifying in the cases in which it has been enforced than was believed possible; especially was this true of the cases of dementia precox. Additional experience has led me to try it in a variety of functional disorders characterized by atonicity either psychic or physical—*e. g.*, in convalescence from the various infections and in migraine.

"My experience includes 30 cases in all. Nine patients were suffering from neurasthenia; 4 of these were men between the ages of twenty-seven and forty-three, and

5 women between twenty-three and forty-eight. In this series the treatment resulted in 8 cures and one cure with recurrence. In a series of 10 patients suffering with psychasthenia, of which 6 were women between the ages of thirty-five and fifty-two and 4 men (one with dipsomania) between thirty-six and fifty, 6 were permanently cured; 2 had a recurrence and 2 patients were improved. In a third group of dementia precox, consisting of one woman, aged twenty-five, and 6 men between twenty-two and thirty-five, 5 patients were permanently cured; in 2 the outcome is as yet unknown. Two women of the hysterical type, both aged twenty-five, were cured. Two patients, one a woman aged twenty-three, suffering from psychasthenia with melancholia, and the other, aged sixty-nine, with recurrent melancholia, were cured.

"Theoretically the method seemed capable of scientific application. Practical results of such application not only warrant its further use, but lead me to look on it as a powerful therapeutic aid that should give as good, if not better, results in the hands of others, if used with intelligent enthusiasm."

DISEASES OF THE BRAIN AND MENINGES.

THE MENINGES.

Serous Meningitis and Other Temporary Organic Brain Diseases as a Cause of Blindness. Muskens and Snellen[7] examined the inmates of an institution for young blind people in order to be able to contribute to the solution of the following problem: What percentage of the blind, taken at large, suffer from atrophy of the optic nerve as a sequel to optic neuritis and choked disc, the original cause (local or general brain disease) being cured or arrested? The following is a summary of the conclusions reached:

1. An unexpectedly large percentage of the pupils in a school for blind boys and girls were found never to have suffered from any ocular trouble, but to have been afflicted in early youth or about the time of puberty

---

(7) Review of Neurol. and Psychiat., May, 1912.

with some brain disease, accompanied by increased intracranial tension, choked disc and, as a sequel, optic atrophy.

2. This high percentage (20 out of 98 pupils) is probably lower than the truth, because all blind children who had given signs of defective mental qualities, fits or paralysis had been refused admission.

3. In these patients arrested cases of tuberculosis, syphilitic and various other new growths may be frequent; the result, however, of the inquiries from the medical attendants of the children (from the general practitioner on the one hand, and the oculist on the other) gives rise to a strong suspicion that more frequently encephalo-meningitis serosa (under which so-called pseudo-tumor and Aztec-skull (*Turm-Schädel*) are to be included), and meningitis serosa migrans, are at the bottom of the condition.

4. The question, whether this form of blindness can be prevented, has been affirmed in the present state of diagnosis and technic by all recent observers for the last 20 years, therein supporting the statements of Sir Victor Horsley. If a prophylatic proceeding be started in the same way as with ophthalmia neonatorum, a similar lowering of the number of the blind may be expected.

5. The palliative operation, wherever there is increased intracranial tension, can be compared with iridectomy in glaucoma and with paracentesis in otitis media. They all aim at saving the function of an important organ, and are harmless operations performed at a period in which most effect may be expected. The best period for palliative trephining is as soon as a choked disc of more than two diopters' swelling does not subside spontaneously in a few weeks. Waiting till vision has suffered must be condemned.

6. Pediatrists and ophthalmologists are most concerned in these rules, since after an inquiry it has been found that children are most prone to suffer.

7. It seems advisable that in the forms for admission to institutions for the blind, some questions should be inserted in view of this cerebral origin of the affliction.

8. Also in the decennial counting of the population special questions should be inserted regarding the blind

citizens, because such an inquiry might elucidate the frequency of this cause of blindness.

Cysticercus of the Brain and Cord and Cysticercus Meningitis, are exhaustively discussed by Kurt Goldstein[8] who reports 5 cases in detail.

Case 1. A man, 62 years old, previously healthy, began having sudden attacks of unconsciousness in the summer of 1904. During the attacks he trembled, had involuntary discharges and sometimes jerked the arms and made movements with the mouth and eyes. He gradually grew dull and the memory became impaired. When observed at the hospital a year later he had a rhythmic tremor in both arms, stronger in the right one, and a slight tremor in the legs. The tremor was increased by motion. On passive motion there was distinct resistance both in the arms and legs, and the patient's attitude was stooping. The knee- and ankle-jerks were increased, but there was no Babinski sign. The speech was strongly nasal and indistinct, as if he were talking with his mouth full. The attacks continued and he occasionally vomited. When again examined in May, 1905, the pupils were unequal and reacted sluggishly to light. The rhythmic tremor persisted.

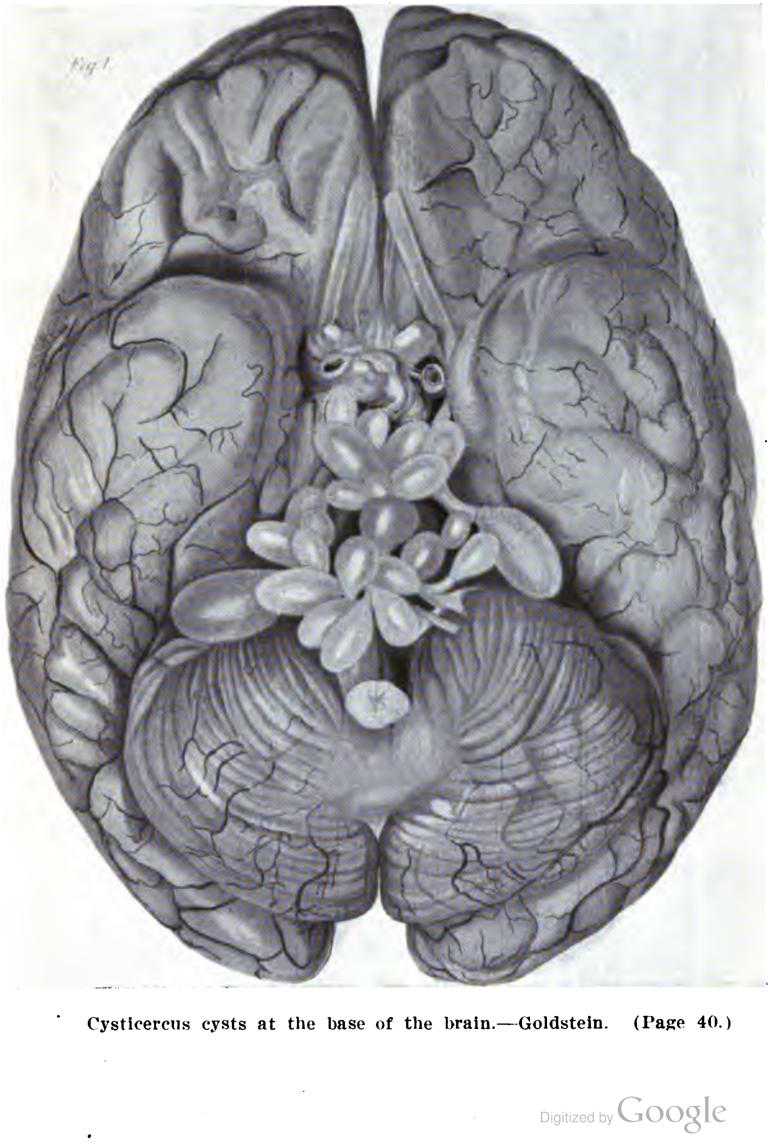
He died a few months later and the most striking finding at the necropsy was a cluster of grape-like cysts at the base of the brain. (See Plate II.) They varied in size from that of a pea to that of a hazel-nut and had thin, translucent walls. The pia matter covering the portion of the brain to which the cysts were attached appeared cloudy and thickened as if infiltrated with a grayish-yellow mass. A chain of small cysts arranged like a string of beads was found between dura and pia along almost the entire length of the spinal cord, and the spinal pia was thickened and infiltrated in many regions. The ventricles of the brain were considerably dilated and the ependyma granular. The fossa Sylvii was the seat of a dense mass of closely-packed cysts which invaded the frontal lobe and also the postcentral convolution. The cysts were of the nature of cysticercus cysts.

A correct diagnosis was not made during life, as the

---

(8) Archiv f. Psychiat., Vol. 49, p. 742.

PLATE II.



Cysticercus cysts at the base of the brain.—Goldstein. (Page 40.)

case was looked upon as one of cerebral arteriosclerosis, and it was thought that the tremor might have been due to this cause or to a complicating true paralysis agitans. There was little variation in the symptoms, while great variability is the rule in cysticercus disease. Tremor and even retropulsion and propulsion have been described, but never such a pronounced picture of paralysis agitans as existed in this case.

Case 2. This case was noteworthy on account of the presence of mental disturbance of the character of typical Korsakow's phychosis. The pupils were sluggish, and optic neuritis was present. The patient had not been an alcoholic. The spinal fluid showed lymphocytosis and a strong "phase 1" globulin reaction, but the Wassermann test was negative. Clinically it was thought most probable that the case was one of tumor, perhaps of the frontal lobe. The chief post-mortem findings were numerous cysts at the base of the brain, marked dilatation of the ventricles and whitish thickening of the pia water.

Case 3 also showed the clinical picture of Korsakow's psychosis characterized by disorientation, marked loss of memory, confabulation and pseudo-reminiscences. There were general muscular tenderness, sluggish pupils and considerable tremor of the whole body. The patient died eight months after the onset. As this patient had used considerable alcohol the most probable clinical diagnosis was true Korsakow's psychosis and cerebral arteriosclerosis. Typical cysticercus cysts of the meninges and internal hydrocephalus were found, and it is interesting that numerous calcified trichinæ were present throughout the muscles, which to some extent may have accounted for the marked muscular tenderness.

Case 4. The patient was a musician 28 years old. As a child he began to have attacks of dizziness, vomiting and headache. His forehead had always been very high, suggesting hydrocephalus. Three years before death the vision and the sexual power began to decrease. Vision was particularly poor during the attacks of headache. He also complained of failing memory. There was marked optic neuritis with beginning atrophy and the visual fields were concentrically narrowed. Exophthalmos was present, the tendon-reflexes were increased and generalized epileptiform convulsions were present. He then improved greatly, even vision becoming almost normal, and the headaches ceased. Finally the convulsions returned and increased in frequency until he had seventy in one day. Lumbar puncture now, as well as at a previous examination, showed increased pressure, lymphocytosis and a positive albumin test. Clinically the case suggested a tumor, most likely either in the third ventricle, frontal lobe, or region of the hypophysis. The *x*-ray showed some widening of the sella turcica, but the diagnosis of serous meningitis was finally made on account of the long period of remission and the history of the symptoms suggesting hydrocephalus in childhood. Cysticercus meningitis was not considered during life, but at the necropsy cysticercus cysts were found at the base of the brain, especially in the vicinity of the infundibulum and chiasma; and between the cerebellum and right temporal lobe there was a cyst of the size of a pigeon's egg. High-grade hydrocephalus was present.

Case 5. A man 52 years old gave the history of having passed a tape-worm at the age of 25 years and of having been subject to headaches all of his life. Eight months before death he developed marked dizziness and later diplopia, failing vision and vomiting. The headache was very severe, particularly in the occipital region. On passive movements of the head he would become dizzy and the pulse became more rapid. The pulse ordinarily ranged between 56 and 65 and was irregular. The urine contained both albumin and sugar. The pupils reacted sluggishly to light, and optic neuritis was present. The tendon reflexes were increased. The spinal fluid showed lymphocytosis and a positive globulin test. The patient died suddenly from respiratory failure. In this case a clinical diagnosis of cysticercus of the fourth ventricle was made and confirmed by necropsy. The fourth ventricle was enormously distended and filled with a cysticercus cyst. The other ventricles were also dilated, and the aqueduct of Sylvius was from ½ to ¾ cm. in diameter.

CEREBRAL LOCALIZATION.

Sensory Disturbances from Cerebral Lesions. One of the most extensive works on sensation ever published is a paper by Henry Head and Gordon Holmes.[9] This paper formed the Croonian lectures before the *Royal College of Physicians* in 1911. The authors examined a large number of selected cases, such as cases of thalamus lesions, and patients with limited lesions of the surface of the brain, mostly cases of successful removal of tumors, and many cases of lesions of the internal capsule.

The authors first take up the grouping of afferent impulses in the *spinal cord and brain-stem.* Their conclusion in regard to the way sensory impulses are carried at the extreme upper end of the spinal cord, is that they pass upward in three main divisions, as follows:

1. The impulses underlying sensations of pain, of heat and of cold are traveling in secondary paths in the opposite half of the spinal cord from that by which they entered.
2. Those impulses upon which depend postural recognition and spacial discrimination still remain uncrossed in the posterior column. They consist of one group arising in the deep afferent system, upon which depends our recognition of posture, passive movement, weight, consistence and vibration. A second group arises in the end-organs of the skin, and these impulses underlie the discrimination of two points applied simultaneously, and the allied appreciation of size, shape and form.
3. Contact sensibility evoked by touch and by pressure may remain entirely undisturbed. Apparently it depends upon impulses which run in a bilateral path, and it is therefore unaffected by a purely unilateral lesion (Rothmann, Petrén, Head and Thompson). This tactile group formed by the union of tactile impulses within the cord carries with it the impulses which underlie the power of recognizing with accuracy the situation of the stimulated spot, and localization therefore remains unaffected.

---

(9) Brain, 1911, Vol. 34, p. 102-255. In briefer form, see the Lancet, Jan. 6, 13 and 20, 1912.

As to the *brain-stem,* a summary of their conclusion follows:

1. The impulses for pain, heat and cold continue to run up in separate secondary paths on the side of the nervous system opposite to that by which they entered. They receive accessions from the regrouped afferent nerves of the head and upper part of the neck. Although these paths are frequently affected together they are independent of one another, and any one of the three qualities of sensation may be dissociated by disease.

2. Lesions of the spinal cord tend to diminish simultaneously all forms of painful sensibility, but with disease of the brain-stem the gross forms of pain and discomfort may pass to consciousness, although the skin is analgesic. This applies not only to painful pressure but to the discomfort produced by excessive heat.

3. The impulses concerned with postural recognition part company with those for spacial discrimination at the posterior column nuclei. Up to this point they have traveled together in the same column of the spinal cord, but as soon as they reach their first synaptic junction they separate. Above the point where they enter secondary paths the power of recognizing position and passive movement can be affected independently of the discrimination of two points and the appreciation of size, shape and form.

4. It would seem as if those elements which underlie the power of localizing the spot touched or pricked become separated off from their associated tactile impulses before they have actually come to an end in the optic thalamus. The long connection of localization with the integrity of tactile sensibility is here broken for the first time.

Next, the sensory disturbances associated with *lesions of the optic thalamus* are considered. It is known that all afferent fibers passing upward from the midbrain end in the optic thalamus, and from the cells of this organ new fibers pass onward to the cortex. They have studied 24 cases in which symptoms and signs justified the diagnosis of a lesion of the optic thalamus.

Thanks to the work of Déjérine, Roussy and others the "optic thalamus syndrome"[1] is now a recognizable

---

(1) See Practical Medicine Series, 1908, Vol. 10, p. 118.

clinical picture. The essential features are as follows:

1. Persistent loss of superficial sensation of one-half of the body and face. This loss to touch, pain and temperature is more or less definite but the loss of deep sensibility is always more pronounced.
2. Slight hemiataxia and more or less complete astereognosis.
3. Acute pains on the same side, persistent, paroxysmal, often intolerable and yielding to no analgesic treatment.
4. Slight hemiplegia which produces no contracture and rapidly passes away.
5. Choreic and athetoid movements in the limbs of the affected side.

Roussy pointed out that of these symptoms only the sensory loss and the pains are directly due to the lesion of the optic thalamus while the other symptoms are produced by the destruction of surrounding parts. It must also be borne in mind that most thalamus lesions are vascular in origin, that neighboring structures are generally more or less involved, and that the distribution necessarily is according to vascular areas and not according to physiologic tracts. Their conclusions in regard to the thalamus are as follows:

No sensory function is so frequently affected as the appreciation of posture and the recognition of passive movements. The amount of this loss varies greatly from a scarcely measurable defect to complete want of recognition of the posture of the limbs on the abnormal half of the body.

Tactile sensibility is frequently diminished, but, excepting in a few cases where all appreciation of contact was destroyed, a threshold could be obtained. It was always possible to show that increasing the strength of the stimulus improved the character of the answers unless the observations were confused by the disagreeable tingling or other accessory sensations.

Localization of the spot touched was defective in half the cases where sensation was sufficiently preserved to carry out accurate tests. This inability to recognize the site of stimulation was equally great whether the patient was pricked or touched. In cases where localization was gravely affected the disagreeable sensation so easily evoked tended to spread widely on the abnormal half of the body. A prick on the hand may cause an extremely painful sensation in the cheek or side, and sometimes the patient simply recognized the stimulus as a change within himself and did not refer the discomfort from which he suffered to the action of any external agent.

Sensibility to heat and cold may show all degrees of change from total loss to a slight increase of the neutral zone. Heat and cold are not dissociated, and if one form of sensation is lost the other will be gravely disturbed. The apparent exceptions arise from a misinterpretation of the sensation evoked on the affected half of the body by high or low temperature.

Not infrequently the compass-test cannot be carried out because of the gross loss of sensation and inability to recognize contact; but whenever the method can be applied a threshold can be worked out, and widening the distance between the points increases the accuracy of the answers.

The power of estimating the relation between two weights is frequently disturbed on the abnormal half of the body. If the appreciation of posture and movement is affected, the patient can no longer recognize the identity or the difference of two weights placed in the unsupported hands. But, so long as tactile sensibility is not diminished, he can still estimate the relation between weights applied one after the other to the same spot, and can recognize the increase or diminution in weights of an object already resting on the hand.

The appreciation of relative size is often disturbed in these cases but with care it is usually possible to demonstrate a difference—threshold. Shape and form in three dimensions are frequently not recognizable on the affected hand, but if tactile sensibility is not grossly affected, the patient usually retains an idea that the object possesses a form, and one may obtain a considerable percentage of right answers.

Vibration of the tuning-fork was recognized by all but three of the patients. In almost every case, however, the length of time during which it was appreciated was shorter and sometimes the rate of vibration was thought to be slower on the affected half of the body.

Roughness, as tested with Graham Brown's esthesiometer, was always recognized, except in 3 cases where the loss of all forms of sensation was extremely severe. Usually the threshold was the same on the two sides, but it was occasionally raised on the affected hand.

The third part of the paper deals with *sensory disturbances produced by lesions of the cerebral cortex.* Each type of sensibility was studied in great detail.

I. TACTILE SENSATION. 1. A cortical lesion may reduce the accuracy of response from the affected part to graduated tactile stimuli. The form assumed by this defective sensibility differs from that produced by lesions at other levels of the nervous system. The affected part may respond to the same graduated hair as the normal part; but this response is irregular and uncertain. Increasing the stimuli may lead to no corresponding improvement, and even the strongest tactile hair may occasionally evoke less certain answers than a hair of much smaller bending strain. Moreover, a touch with the unweighted esthesiometer may be as effective at one moment as the same instrument weighted with 30 gm. at another. In such cases no tactile threshold can be any longer obtained.

2. This irregularity of response is associated with persistance of the tactile sensation and a tendency to hallucinations of touch. Where the sensory defect is not sufficiently gross to abolish the threshold, persistence, irregularity of response, and a tendency to hallucinate may still disturb the record.

3. In all cases where tactile sensibility is affected, whether a threshold can be obtained or not, fatigue is induced with unusual facility. Although the patient may cease to respond to tactile stimuli over the affected part in consequence of fatigue, his answers will remain as good as before from the normal parts. Over hairless parts stimulation with cotton-wool may produce a sensation which seems "less plain" to the patient, and his answers may show the same inconstancy

so evident when he is tested with graduated tactile stimuli.

II. PAINFUL STIMULI. A purely cortical lesion leads to no change in the threshold to measurable painful or uncomfortable stimuli. Nor does the patient express greater dislike to these stimuli on one side than on the other. A prick may be said to be "plainer" or "sharper" on the normal than on the affected side, but this is due to a defective appreciation of the pointed nature of the stimulus and bears no direct relation to the painfulness of the sensation evoked.

III. TEMPERATURE SENSE. The appreciation of heat and cold was not affected in a considerable number of cortical lesions. When disturbed the following changes were found:

1. The neutral zone, within which the stimulus was said to be neither hot nor cold, was considerably enlarged in comparison with that observed on similar normal parts of the same patient.

2. The patient complained that, although he recognized correctly the nature of the stimulus, it seemed "less plain" than over normal parts. His answers were less constant and less certain; a temperature recognized without difficulty at one time seemed doubtful at another.

3. The power of discriminating the relative coolness of two cold stimuli or the relative warmth of two hot tubes may be diminished. Thus 20° C. may be said to be the same as ice, and 40° C. may seem as warm as, or even warmer than, 48° C. The faculty of appreciating the relation to one another of two temperatures on the same side of the scale is disturbed.

IV. THE APPRECIATION OF POSTURE AND OF PASSIVE MOVEMENT. 1. Cortical lesions most frequently disturb the recognition of posture and of passive movements. Whenever sensation is in any way affected in consequence of a cortical lesion these two functions suffer.

2. In all cases the disturbance in the faculty of recognizing posture and passive movements was greater towards the peripheral parts of the affected limb.

3. When a patient with unilateral disturbance of

these faculties attempts to point to some part of his body, defective knowledge of its position causes greater error than want of recognition of posture and movement in the hand with which he points.

4. When testing the patient's power of appreciating passive movement, the answers are frequently uncertain, and hallucinations of movement may occur; yet the patient may be remarkably consistent and accurate when normal parts are tested.

V. LOCALIZATION. By this we understand the power of recognizing correctly the spot that has been stimulated.

1. The power of localizing the stimulated spot is not commonly lost as a consequence of cortical lesions.

2. This faculty is independent of the power of recognizing the position of the affected limb; appreciation of posture may be lost, although localization is not in any way disturbed.

3. If the power of localization is lost, the patient will be unable to recognize not only the position of a spot touched but also the position of a prick.

4. When localization is defective the patient does not habitually localize in a particular direction, but ceases to be certain where he has been touched or pricked.

VI. THE COMPASS-TEST. 1. A cortical lesion may destroy the power of discriminating two compass-points, both when applied simultaneously and successively. If this is the case, no threshold can be obtained for either form of the test; increasing the distance between the points does not constantly improve the accuracy of the answers.

2. This disturbance is not dependent on changes in tactile appreciation, for it can be demonstrated equally well with two painful as well as two tactile stimuli.

3. The condition of tactile sensibility and the accuracy of the simultaneous compass-test are closely associated; a raised tactile threshold is usually accompanied by a raised threshold for the appreciation of two points simultaneously applied.

4. Should the power be preserved of recognizing two points when the compasses are applied consecu-

tively, localization will be found to be intact. The patient's appreciation of the two points when they are separated by an interval of time is due to the recognition of the separate locality of the two spots touched.

VII. THE APPRECIATION OF WEIGHT. 1. The power of estimating the relative weight of two objects of the same size and shape is readily disturbed by cortical lesions.

2. Though the patient retains sensations of contact when the weight is placed in his hand, all power of recognizing the relative heaviness of the object has disappeared.

3. This faculty is equally disturbed in most cases whether the weights are placed on the supported or the unsupported hand.

VIII. APPRECIATION OF SIZE, SHAPE AND FORM IN THREE DIMENSIONS. The correct appreciation of size, shape and form does not depend entirely on the recognition of posture, for it was perfectly retained in two cases although postural recognition and the appreciation of passive movement were gravely affected. But in every case where the sense of posture was affected and the authors were unable at the same time to obtain a tactile threshold, all recognition of size, shape and form was abolished. Not that such recognition depends directly upon the state of tactile sensibility, but the combined affection of the sense of posture and loss of the tactile threshold indicate simply that the defect produced by the lesion has reached a sufficient grade to destroy the power of recognizing identity and difference. The appreciation of size, shape and form, therefore, suffers alike in all tests which imply a relation.

IX. ROUGHNESS AND TEXTURE. The power of appreciating roughness is ordinarily retained, but that of recognizing texture is usually lost on the affected side so that cotton, silk and velvet feel alike.

X. VIBRATION. While a lesion at lower levels of the nervous system frequently destroys all power of appreciating the vibration of a tuning-fork, this is not the case with a cortical lesion. There may, however, be quantitative disturbance.

In the fourth and last part of the paper the authors attempt to give a general analysis of their results and to present a coherent account of the mechanism of sensation. To be fully understood this must be read in the original. Suffice it to quote some of their statements regarding the characteristics of sensory disturbances of cortical origin:

"Evidently a lesion of the cortex produces an effect upon sensation of a different order from that found at any lower level of the nervous system. All those defects which may occur when a normal person is under examination with psycho-physical tests are present in a grossly exaggerated form. Those defects which we ordinarily attribute to want of attention are raised to a degree unknown in the normal human being. But the patient's power of general attention has not been lowered in such cases as we have cited in this paper. Records from the limbs of the sound side, show that the phenomena of defective attention are confined to the abnormal parts. A portion of the patient has, as it were, become untrustworthy.

"To sum up, we believe that the cerebral cortex is the organ by which we are able to focus attention upon the changes evoked by sensory impulses. A pure cortical lesion which is not advancing or causing periodic discharges will change the sensibility of the affected parts in such a way that the patient's answers appear to be untrustworthy. Such diminished power of attention makes the estimation of a threshold in many cases impossible. Uncertainty of response destroys all power of comparing one set of impressions with another and so prevents discrimination. But in addition to its function as an organ of local attention the sensory cortex is also the storehouse of past impressions. These may rise into consciousness as images, but more often, as in the care of special impressions, remain outside central consciousness. Here they form organized models of ourselves which may be termed 'schemata.' Such schemata modify the impression produced by incoming sensory impulses in such a way that the final sensations of position and of locality rise into consciousness charged with a relation to something that has happened before. Destruction of such schemata by a lesion of the cortex renders impossible all recognition of posture or the locality of a stimulated spot in the affected part of the body."

Hallucination of Smell may occur in lesions of the temporal lobes (gyrus hippocampi, uncus, cornu Ammonis) while loss of smell may be produced by affections of the olfactory bulb or tracts or by destructive lesions of the centers. According to Kutzinski[2] hallucinations of smell are rare in brain tumor and altogether there are only 13 cases of this kind reported. He reports the case of a man 36 years old who had a few epileptiform seizures preceded by an olfactory aura. The attacks gradually increased in number and he developed symptoms of increased brain pressure, such as choked disc and occasional stupor and confusion. The necropsy revealed a glioma on the right side in the vicinity of the basal ganglia.

Disturbances of Taste in Tumors of the Posterior Cranial Fossa are discussed by B. Scholz[3] who first of all calls attention to the disagreement between anatomists and physiologists on the one hand and neurologists on the other regarding the course taken by the taste impulses. Both sides agree that the impulses may travel from the tongue to the lingual nerve and the chorda tympani and may reach the medulla through the glosso-pharyngeal and trigeminal nerves, but there is wide divergence as to the importance of the facial nerve. Anatomists and physiologists consider the portio intermedia of the facial nerve, the nerve of Wrisberg, the nerve of taste; while practical neurologists, following in the footsteps of Erb, look upon the trigeminal or fifth nerve and the glosso-pharyngeal (ninth nerve) as the nerves of taste. The author agrees with the anatomists and physiologists.

Histologic Examination of the Brain During Life by Means of Brain Puncture. O. Foerster[4] makes a report of numerous cases of various diffuse brain diseases in which small pieces of brain were secured by puncture and subjected to histologic examination. He

---

(2) Med. Klin., No. 10, 1912.
(3) Mitt. aus den Grenzgeb., Vol. 23.
(4) Berlin. klin. Wochenschr., May 20, 1912.

has made such puncture in 16 cases and always over the right frontal lobe. The skin is disinfected with tincture of iodin and frozen with an ethyl chlorid spray. A drill hole is made and then the puncture is performed. In 4 cases of paretic dementia the characteristic plasma-cell infiltration around the vessels was readily seen. In 3 cases which presented symptoms suggestive of the same disease, the pieces removed failed to show the characteristic changes, but showed the histologic picture of arteriosclerotic brain disease. In 5 cases of multiple sclerosis the diagnosis also could be made by this method by means of the proliferation of glia, especially the increase of glia cells along the vessels. The author considers the method of particular value in the difficult differentiation between paretic dementia and syphilitic pseudoparesis in both of which the Wassermann tests and examination of the spinal fluid for lymphocytes and globulin are likely to be positive and hence of no differential value. The same is true when a patient suffering from tabes develops mental symptoms and the important question arises whether paretic dementia is present. Here also the ordinary laboratory tests often fail to enlighten us.

[This method is not likely to be adopted to any extent outside of the large clinics of continental Europe. Similar harpooning of the liver and spleen has also been practiced and often yielded satisfactory results as far as diagnosis is concerned, but that the method is not without danger has been pointed out by Chiari and other pathologists who had opportunity to examine the site of the puncture on many occasions. Ed.]

Dominating Right Hemisphere in Right-Handed Person. Kurt Mendel[5] reports a case which he considers the first one on record to show in a convincing manner that the right hemisphere in a right-handed person may be the dominating one and may contain the speech centers. The patient was a woman 42 years old, always right-handed and in whose family every member was right-handed. She had developed a heart lesion and finally had a stroke of left hemiplegia in which she completely lost her speech. She could not speak a

---

(5) Neurolog. Centralbl., Feb. 1, 1912.

single word, but understood written and spoken language and could write both spontaneously and at dictation. She improved considerably in the next few months, but died 8 months after the stroke. The necropsy revealed mitral stenosis, thrombosis in the left auricle, healed infarcts in the spleen and embolism of the right middle cerebral artery proximal to the bifurcation. The resulting area of softening had completely destroyed the third frontal convolution and the island of Reil and, partly, the first temporal convolution. The left hemisphere was absolutely normal, and careful examination of the vessels showed no evidence of embolism or arteriosclerosis.

Of cases in the literature Mendel classes 7 as doubtful and one as "almost certain," namely, one reported by Lewandowsky.[6] The patient was a right-handed man 55 years old. He gradually developed paralysis of the left arm and leg, asphasia and left hemianopsia. He showed sensory aphasia, slight paraphasia, pronounced alexia, almost complete agraphia and distinct motor apraxia of the right hand. At the necropsy a glioma was found in the interior of the right hemisphere extending from the occipital lobe forward into the internal capsule. The left hemisphere was entirely free from tumor.

In this connection the author recalls the very interesting and, so far, not duplicated case reported by Charcot and Pitres in 1894 of a patient with left hemiplegia and aphasia and softening of the left hemisphere. The reason for having the softening and paralysis on the same side was failure of decussation of the pyramids in the medulla.

In the discussion of Mendel's paper Jacobsohn reported an interesting observation which shows that most painstaking histologic examination is necessary to guard against erroneous conclusions. A young patient developed suppuration in the right ear followed by hemiplegia and sensory aphasia. Then the mental confusion, stupor and fever increased and the left ear also began to discharge pus. On account of the sensory aphasia and right hemiplegia occurring in a right-handed

---

(6) Zeitschr. f. d. ges, Neurol, 1911,

person a diagnosis was made of abcess of the left temporal lobe, and operation performed but nothing was found. The patient soon died and at the necropsy the lesion was found to involve the right temporal lobe and right third frontal convolution. There was no gross lesion in the left hemisphere, but on microscopic examination slight inflammatory changes were also found in the first left temporal convolution.

Cerebral Spastic Paralysis.

Rhizotomy (Section of Posterior Roots) in the Treatment of Spastic Cerebral Paralysis. This subject was discussed in the *Practical Medicine Series*, 1911, Vol. 10, p. 168, and 1910, Vol. 10, p. 72. More recently L. Pierce Clark and Alfred S. Taylor[7] analyze the results on the basis of 41 cases of Little's disease of which records now are available. Küttner heads the list with 10 cases and the authors themselves have 5 cases. They point out that the disability of the limbs in these cases is due to two causes, namely weakness and spasticity, and it is the latter which is influenced by the operation. The ideal age for the operation is between the fourth and seventh year, when the child has acquired the principles of balancing and walking and is too young to have acquired marked trophic disturbance and deformities. In the presence of deformities, it is often difficult to decide whether tenotomies should be undertaken before or after the root section and this can often only be decided by co-operation between surgeon and orthopedist.

Foerster has carefully worked out a table showing which roots should be resected for the correction of spasticity of the muscles of the lower extremity. This table, of course, is based on our knowledge of the cord segments and anterior roots from which fibers pass to innervate the various muscles. Thus, in a case of contracture of the adductors of the thigh, the second to the fourth or possibly the fifth lumbar roots should be resected. The flexors of the leg, the hamstring muscles, are represented in the fifth lumbar and first and

---

(7) N. Y. Med. Jour., Apr. 13, 1912.

second sacral segments and the same is true of the plantar flexors of the foot, that is of the calf musculature. For spastic paralysis of the arm, the best results generally follow section of the fourth, fifth, sixth, seventh and eighth cervical and first dorsal roots.

There is apt to be some pain and tenderness for five or six weeks after the operation. The position of the legs rapidly becomes normal. Anesthesia and trophic disturbances are usually avoided if not more than three consecutive roots are cut. Temporary retention of urine was noted in a few of Foerster's cases but not in any of the authors' cases.

"The practical benefit accruing in most cases from return of power to execute isolated movements is best seen in the patients' gait. They can usually flex the foot both ways to full amplitude, flex and extend the legs normally, raise the limbs high up, abduct and adduct and rotate in and outwardly. The apparent gain in power after operation is but the releasing of the corticogenic excitability of certain muscles bound down previously by spasticity. It hardly seems probable, as Foerster contends, that some voluntary motion is added, for the first time, after operation. With the removal of contractures and restoration of an approximately normal breadth of excursion of movements of limb segments, and the elimination of the very perturbing defensive flexor reflex, and removal of associated movements (associated with voluntary efforts), and, finally, last but not least, with the return of voluntary motion, we have a basis for developing *standing* and *walking* in our diplegic patients. Even at this point the child must go through a very elaborate education in using the extremities. At first it collapses. It first must acquire the act of holding the knees extended, and second, of raising the pelvis and trunk on the legs. First the patients stand with bilateral, then unilateral support, then they learn to walk with a cane, and finally to go alone.

"The after-treatment must be prolonged. There can be no doubt that the after-care and training of cases treated by dorsal root section is the most important part of the whole procedure as far as the enduring results go, once the root section is made. Even while the little patients are in bed, the first few days after operation, diligent daily exercises of the legs should be given. These exercises should be undertaken several times daily, even though some of the pain on movement still remains. The painful soreness is aided by the treatment, too. If the limbs do not return to the normal rest position after the operation, removable plaster sheaths should be applied. This treatment should gain the maximum extension, abduction and external rotation in the hip-joint, maximum extension of the knees, dorsal flexion or medium position of the foot, etc. In the slighter grade of post-operative deformity sand-bags may be used. All this apparatus should be used while the patient is bed-ridden and while he is not undergoing active treatment in exercises, and especially during the night. Foerster considers these sheaths or external corrections of abnormal positions necessary during several years. He recommends in extreme flexure-contractions at the hip, that a cushion be slipped underneath the sacrum of the recumbent patient.

"The form of passive exercises in detail for the patient during the bed-ridden period may be said to consist in having the patient make, 1, resistive hip-extension and flexion and passive stretching of hip extensors; 2, resistive hip-abduction and passive stretching of hip abductors; 3, resistive outward hip-rotation; 4, passive flexion and extension of the foot and resistive movement in the same manner; 5, resistive foot-inversion; 6, with bent knees resistive knee-separation; 7, sitting hip- and knee-flexion and extension; 8, co-ordination of standing by placing the foot forward and sideways on command; 9, hands on hips and practicing rising and sitting on command; 10, sitting with feet on the floor and practicing foot in dorsal flexion, each foot separately. In brief, the foregoing may be considered as some of the literal and specific trainings these cases may follow through."

As to the effect on the reflexes, the authors have found the facts to be as follows:

1. Ankle-clonus is usually abolished at once, permanently in all cases. Foerster believes that either S_1

or S_2 can maintain the ankle-clonus as in the very few cases where ankle-clonus has persisted, one or the other of these roots was not resected.

2. Patellar reflexes were abolished in all cases of Foerster's but not in others, notably our own and those of Göbell's.
3. Babinski's reflex persists in all reported cases except in one reported by Kotzenberg.
4. Oppenheim's tibial reflex persists in some cases and disappears in others.
5. The Achilles reflex is lost or markedly diminished in all the typical resections, that is, L_2 L_3 L_5 and S_2. In two cases in which ankle-clonus persisted the Achilles reflex was also present. The effect, however, of radiculotomy is of theoretic rather than of practical interest.

The abolition, or at least marked reduction, of the defensive flexion reflex in all cases, is absolutely necessary and has been obtained in nearly all cases. Its marked continuance indicates a failure in the operation. A pathologic increase of this reflex causes the irrepressible flexor-cramps in diplegics. They are extremely annoying and often painful to the little patients. If no other causes were in evidence, this one alone would prevent walking and standing in the vast majority of cases.

Partial Resection of Motor Nerves in Athetosis and Other Motor Disturbances have been practiced with good results by Medea and Bassi[8] of Milan. In a case of athetosis in which involuntary movements of the latissimus dorsi played the principal rôle the result of partial resection of the nerve supplying this muscle was extremely successful in that the patient retained the power of innervating the muscle normally and yet the abnormal movements were largely checked. Similarly in a case of excessive pronation of the hand they succeeded by means of mechanical irritation of the exposed median nerve at the elbow to isolate a small bundle of fibers, irritation of which resulted in the isolated movement of pronation. By resecting half of this bundle for a distance of two inches the abnormal

---

(8) Neurolog. Centralbl., June 1, 1912.

movements were greatly lessened and the power of normal motion retained.

CEREBRAL SYPHILIS.

Cerebral Syphilis and Trauma. Zipperling[9] reports a case and sifts the evidence which has caused many experienced observers to believe that a trauma may not infrequently precipitate symptoms of cerebral involvement in a syphilitic subject. Thus, Oppenheim in his text-book says: "It is not unusual to observe that the first symptoms of brain syphilis appear immediately after an injury to the head." Nonne, on the other hand, with his enormous experience, has become convinced that one must be careful not to overestimate the influence of trauma.

The author's patient was a man 26 years old who gave a history of chancre three years before sustaining an injury. A bag weighing about 50 pounds fell on his occiput. He became unconscious and after eight days at the hospital, where no bone injury could be demonstrated, he returned to work but found that he easily became dizzy, so he stayed at home a few days. When he again returned to work he was very forgetful and could not hold any position. He also had frequent brief dizzy attacks. There were no findings on neurologic examination, but the Wassermann test was positive both with the blood and spinal fluid, and lymphocytosis and globulin were present. He improved greatly on mixed treatment. The essential fact in the case was that the man had showed no cerebral symptoms whatever prior to the injury.

The author accepts very few of the reported similar cases in the literature as genuine. A fairly convincing one is reported by F. Dreyer.[1] A bricklayer, six years after his chancre, was injured by a brick which fell on his occiput. This was followed by severe headaches and dizzy attacks and deviation of the tongue to the right. He recovered after specific treatment. Many other cases are suspicious because the history of the

---

(9) Neurol. Centralbl., Dec. 1, 1911.
(1) Inaugural Dissert., Göttingen, 1899.

injury would lead one to suspect that dizziness or other weakness caused by the syphilis was the cause of the injury, so that we are really dealing with traumas caused by cerebral syphilis and not the reverse. The same is true of the reported cases of tabes and general paresis following injury. The problem becomes more complicated by the fact that the work of Quincke and others has shown that head injuries not rarely give rise to a form of serous meningitis with increase in pressure of the cerebrospinal fluid. In the author's case quoted above, it is possible that the injury produced a serous meningitis responsible for the increase in pressure of the cerebrospinal fluid, and that thereby the meninges became more susceptible to the action of the syphilitic virus. On the whole, however, the author considers it very questionable whether a head injury will cause syphilis to become localized in the brain if the brain be not already affected.

The Nonne-Apelt "Phase 1" Test for Globulin in the Spinal Fluid, by the addition of equal parts of a neutral saturated solution of ammonium sulphate which causes a white turbidity, has been applied in many cases of various forms of nervous disease by L. Beltz[2] who formulates the following conclusions:

1. The Nonne-Apelt reaction is of value in the solution of the question whether an obscure case of disease of the central nervous system is of syphilitic origin.
2. It is always positive in paretic dementia.
3. It is nearly always positive in tabes.
4. If cerebrospinal syphilis is suspected a positive test favors this diagnosis while a negative one does not exclude it.
5. Inflammatory disease of the meninges, both tuberculous and suppurative, generally give a positive reaction.
6. Faintly positive reactions may be obtained in the meningeal irritation of pneumonia, acute leukemia and other diseases as well as immediately after an epileptic attack.
7. A certain irregularity in the results of the vari-

---

(2) Deutsche Zeitschr. f. Nervenheilk., Vol. 43, p. 63.

ous tests with the spinal fluid is characteristic of cerebrospinal syphilis.

8. In tabes the Wassermann test with the spinal fluid is generally negative.

9. A strongly positive globulin reaction in the absence of lymphocytosis of the spinal fluid should direct suspicion to the existence of compression of the cord.

The Spirocheta Pallida, judging from the scarcity of reports, is not readily found in the nervous system. Phillips and Glynn[4] discuss the various methods of examining for this organism. The appearance of the spirochetes in sections from the nervous system is seen in Plates III and IV from Strassman's[5] work in which he describes a case of cerebral syphilis with onset of nervous symptoms a few months after the chancre. This case will be found related in the *Practical Medicine Series*, 1911, Vol. X, p. 98.

A New Test with the Spinal Fluid in Syphilitic Nervous Diseases by Means of Gold Chlorid. Extremely promising and noteworthy work on the cerebrospinal fluid is in progress at Wechselmann's clinic in the *Rudolf Virchow Hospital* in Berlin. It is here that the most extensive and minute clinical studies on salvarsan have been made, so the available clinical material is enormous. Carl Lange,[6] an assistant in this clinic, has made a thorough study of the behavior of the spinal fluid with the previously known tests for albuminous substances, and proposes a method which opens up possibilities of unexpected refinement in diagnosis. He first observed that in certain cases, especially in early syphilis when the "phase 1" globulin test was negative, *i. e.*, no cloudiness was obtained within three minutes by mixing the fluid with equal parts of saturated solution of ammonium sulphate, a slight precipitate would form on standing for 24 hours. It appears that entirely normal fluids remain perfectly clear even for this long period. This phenomenon led the author to suspect that the ammonium sulphate method was not sufficiently delicate in early syphilis and he went to work to find something better.

---

(4) Brit. Med. Jour., Nov. 11, 1911.
(5) Deutsche Zeitschr. f. Nervenheilk., Vol. 40, p. 387.
(6) Zeitschr. f. Chemotherap. u. verwandte Gebiete, Vol. 1, p. 44.

Google

This search led to many interesting side observations before the new test was perfected. If the fluid is half saturated with ammonium sulphate, centrifuged, and the resulting clear fluid saturated with the dry salt a marked cloudiness may be obtained in pathologic fluids and a slight one in normal fluids. This may give a positive result when "phase 1" is negative.

The reaction of the fluid is a factor which has not received sufficient attention. It is distinctly alkaline and persistently so, for if hydrochloric acid is titrated until the previously red phenolphthalein indicator has lost its color, it will again turn red on standing a few hours, and this may be repeated seven or eight times. The degree of alkalinity of the fluid may also be an important factor in connection with the Wassermann test as high alkalinity tends to inhibit the reaction, which may be one reason why it is more frequently negative in the spinal fluid than in the less alkaline blood serum.

As to the "phase 1" reaction, it seems clear that it is a group reaction of various albuminous bodies and is capable of further and useful subdivisions. Lange began to experiment with the "colloidal gold," produced by Zsigmondy by the reduction of gold chlorid in the presence of an alkali. The resulting solution is absolutely clear and of a beautiful purple color. When electrolytes, such as salt solution, are added the fluid at first turns blue and a bluish black mass is deposited, and in a few hours the fluid is clear as water.

Zsigmondy made the important discovery that the various albumins show quantitative difference, so each one has a certain "gold coefficient," by which is meant the number of milligrams of the albuminous body which will protect 5 c.c. of the colloidal gold against 0.5 c.c. of 10-per-cent. sodium chlorid solution. In this way it may be determined whether a certain albumin exists entirely alone, and if so, at what concentration. It is curious that while proto- and heteroalbumoses act as protectors, the deutero-albumoses play the rôle of electrolytes, even in the presence of other albuminous bodies which usually prevent the separation out of the gold. Lange tried to discover whether by this method differences between normal and syphilitic blood-sera could be obtained but got no results of practical importance.

With the spinal fluid, on the other hand, results of practical importance were obtained in the following manner. The spinal fluid is first mixed with 10 parts of 0.4 per cent. sodium chlorid solution. There must be no admixture of blood. Then a series of dilutions ranging from 1 in 10 to about 1 in 40,000 are made in numerous test-tubes by adding sodium chlorid solution. First 1 c.c. of the salt solution is put in each tube. Tube 1 contains the original 1 c.c of the fluid and 10 c.c. of 0.4 per cent. salt solution; tube 2, 1 c.c. of tube 1 plus 1 c.c. of salt solution, or a dilution of 1 to 20; tube 3, 1 c.c. of tube 2 plus 1 c.c. of salt solution, or a dilution of 1 to 80, etc. The twelfth tube would have a dilution of 1 to 20,480 and 12 tubes are shown in the author's illustration of the reaction. Then 5 c.c. of the gold solution are added to each tube and any change in color is noted. Normally none occurs, not even after standing 24 hours. The tubes should be left over night or at least several hours before the results are recorded.

With pathologic fluids the gold becomes separated out, which is indicated by a change of the purple color of the solution to reddish blue, lilac, dark blue, light blue or whitish blue. The most complete change consists in the formation of a blue sediment with a supernatant fluid clear as water. It is noteworthy that the greatest change is not usually in the first tube with the greatest concentration of fluid but in the second, third or fourth. For instance, in a case of beginning tabes the first tube (1 to 10 dilution) remains unchanged, while the greatest change is in the third one (1 to 80 dilution) which becomes clear, with the formation of a precipitate. After the eighth or ninth tube the fluid again remains unchanged. The most pronounced reactions are generally obtained in cases of cerebral syphilis, tabes and paretic dementia in which the spinal fluid gives a positive Wassermann test. The author is convinced that while a normal fluid may give a faint reaction, a strong reaction is always indicative of disease of the central nervous system. By his cytologic and globulin examinations he has become convinced that some degree of involvement of the nervous system in early syphilis is very much more frequent than is generally supposed.

The test promises to be of great value in differential diagnosis, not only because it appears to be the most delicate test yet devised for the demonstration of a slight change in the central nervous system but also because different reactions are obtained in the various diseases present. Thus, the colors and degrees of dilution most affected are different in suppurative meningitis, tabes, paretic dementia and ordinary syphilis. It will of course require more extensive investigation to determine whether it is possible to construct a certain formula for each disease according to the reactions obtained. So far the author has applied the test in over 100 cases of various forms of nervous disease.

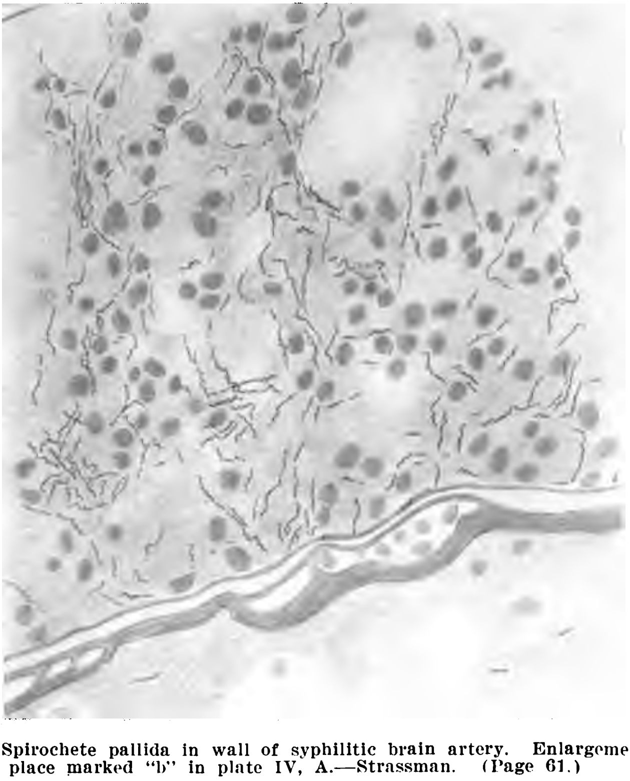
[We will not enter into further details at present but advise our readers to watch for further publications by Lange or other investigators. As the most painstaking technic in the making of reagents, the cleaning of glassware and in the obtaining of the spinal fluid are absolutely necessary it is probable that we shall hear of very contradictory results according to the care in the technic used.—Ed.]

Salvarsan in the Treatment of the Syphilitic Diseases of the Nervous System was one of the subjects discussed at the 1911 meeting of the Society of German Neurologists. The opening paper was read by Nonne[7] of Hamburg who began with a discussion of the value of the Wassermann and other laboratory tests as guides to the treatment. He believes that the Wassermann reaction must be looked upon as a symptom of syphilis, as a sort of metabolic anomaly produced by the liberation of definite chemical substances on the part of the blood-cells. As to the mercurial treatment, on which Nonne lays great stress, after trying all methods of administration he finds inunctions the most satisfactory in tabes. The danger he considers almost negligible. Thus in 28 years' experience with an enormous material he has never seen a case of mercurial

---

(7) Deutsche Zeitschr. f. Nervenheilk., 1912, Vol. 43, p. 166-336.

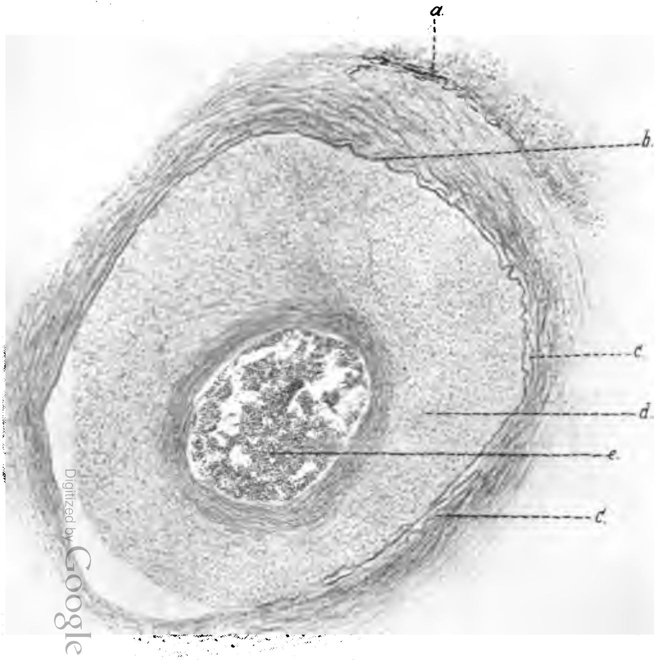
PLATE III.



Spirochete pallida in wall of syphilitic brain artery. Enlargement of place marked "b" in plate IV, A.—Strassman. (Page 61.)

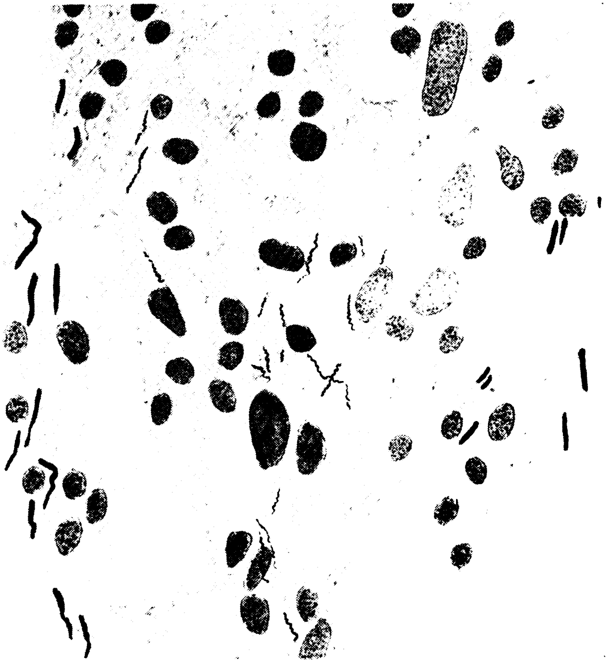
PLATE IV.

A.



cerebellar artery in case of brain syphilis.—Strassman.

B.



Spirochete pallida in sheath of hypoglossal nerve.—Strassman.
(Page 61.)

neuritis. He does not approve of the dictum that the patient should be treated with mercury until the Wassermann test becomes negative, and believes that those who made the demand did not possess sufficient knowledge of the factors involved.

On the basis of the study of the lecithin metabolism by Peritz and Glikin[8] Nonne recommends the administration of lecithin preparations during the antisyphilitic course in order to prevent "lecithin starvation" to which there seems to be a tendency in syphilis. To do this in a rational manner frequent examination of the blood and stools ought to be made, which is a formidable undertaking.

As to the justification for giving salvarsan in the syphilitic and parasyphilitic nervous diseases, Nonne first takes up the question of the possible dangers. He estimates that up to the time of this meeting about 100,000 cases have been treated with this remedy and only 18 deaths attributable to it are on record. A critical review discloses that in 7 of these cases the heart or aorta was diseased, that in 2 cases acid solutions were injected intravenously, that one case showed multiple degenerations of the heart, liver and kidneys, and another a hypoplastic heart and general debility. When we consider the great frequency of aortic and cardiac disease in syphilitics with or without tabes we must assume that a very large number of those treated had disease of these organs, and as the deaths have been so few it must be concluded that salvarsan cannot be very harmful to the heart and aorta.

Nonne does not consider aortitis alone a contra-indication; in fact he has treated several cases of known aortic aneurysm on a syphilitic basis with this drug. There are also numerous cases on record of good results following salvarsan administration in angina pectoris on a syphilitic basis, but without myocarditis. He considers the combination of syphilitic aortitis, coronary sclerosis and myocarditis as a contra-indication to salvarsan. Cases of embolic pneumonia and pleuritis following intramuscular injections he does not charge up against the drug, and reminds us that during 25

---

(8) Deutsche med. Wochenschr., 1911.

years 80 cases were published of death from injections of gray oil. Nonne has treated cases complicated by severe chronic alcoholism with salvarsan without unfavorable consequences. In short he believes that with rational and technically correct salvarsan therapy there is no organotropic effect on the heart, kidneys, stomach or intestines.

On the other hand we cannot be nearly as certain about the harmlessness of salvarsan as far as the nervous system is concerned. The peripheral nerves often become affected, and recently there have been several cases of "acute brain swelling" and hemorrhagic encephalitis after salvarsan treatment.

Nonne is certain that acid solutions should be absolutely prohibited and speaks strongly in favor of the intravenous method of administration as much safer. It has also been shown, particularly in Bornstein's work, that the excretion following intravenous injection may last 1 to 3 months. Another objection which has been raised against the intravenous method, namely, that it can hardly be carried out in a physician's office, can now be set aside as experience has abundantly taught us that one is no more justified in giving these injections at the office than one is in making lumbar puncture there. Nonne adds that the latter procedure was often carried out in offices and dispensaries without subsequent rest in bed until more experience had made us wiser.

He calls attention to the great discrepancies in the reports of various authors as to whether a previously positive Wassermann test becomes negative after salvarsan treatment. The figures vary from 90 per cent. to 4 per cent. and even less. Nonne considers it settled that the influence of salvarsan in changing the Wassermann reaction to negative is less than its effect on the other manifestations of syphilis.

One of the most important tasks in the future will be to collect material to answer the momentous question as to whether early salvarsan treatment will protect the patient against syphilitic disease of the nervous system later in life. Our present knowledge does not enable us to give an opinion.

The author then takes up the question of the so-called nerve recurrences concerning which a lively battle is still being waged. Those who, with Finger, believe that we are dealing with a toxic salvarsan action advance the following points:

1. The present great frequency of peripheral nerve affections compared with their previous relative infrequency. This is also true of multiple neuritis.
2. There is generally only a brief interval between the administration of salvarsan and the onset of the nerve affection.
3. The nervous symptoms often disappear spontaneously and in other cases do not disappear on antisyphilitic treatment.
4. The Wassermann reaction is often negative.
5. The symptoms vary in intensity.

From the other camp which looks upon the nerve recurrences as syphilitic manifestations, with Benario as the foremost champion, the following points are brought out:

1. The symptoms which constitute the nerve recurrences are themselves of occasional occurrence in early syphilis.
2. Variation in intensity is characteristic of the symptoms of certain forms of brain syphilis.
3. Most cases of nerve recurrences recover on antisyphilitic treatment, and there have been cases where this treatment was delayed and recovery was not obtained before the treatment was started. It is of particular interest that in many cases continuation of the salvarsan treatment has brought about recovery where mercury failed.
4. The fact that the symptoms often occur early during the secondary stage when the dispersion of the spirochetes is particularly great.
5. In most cases the interval between the administration of salvarsan and the appearance of the symptom is too long, often months, to admit of the toxic effect of salvarsan playing a rôle.
6. The symptoms in question have never been observed in patients who were treated with salvarsan

for diseases other than syphilis such as leprosy, malaria, relapsing fever, Vincent's angina.

7. The nature of the process is an inflammatory one, not one of chronic degeneration as seen in arsenical poisoning.

8. That the Wassermann reaction is often negative does not appear strange to those who are familiar with the behavior of this reaction. It would only be strange if the test with spinal fluid were negative when a large quantity is used.

9. Mercurial neuritis also occurs, but that is no reason for rejecting mercury.

Most authorities yield to the force of the arguments for the last-named view and agree with Benario. Nonne himself has seen two cases of nerve recurrence among 200 cases treated, both tabetics, one of whom developed a circumflex neuritis causing paralysis of the deltoid, and the other an ocular paralysis. The results of systematic lumbar punctures, particularly Assmann's[9] extensive experiences, also confirm the "meningitic-syphilitic" view. Nonne considers it logical to treat these nerve recurrences with antisyphilitic remedies.

The Therapeutic Results with Salvarsan are carefully analyzed by Nonne in great detail and his final conclusions are as follows:

1. Salvarsan often has a profound effect on true syphilis of the central nervous system.
2. A "sterilisatio magna" rarely if ever is obtained with doses now used.
3. Intensive treatment of primary and secondary syphilis with medium or large doses of salvarsan may be of danger to the nervous system.
4. Treatment of tabes and paretic dementia with salvarsan is not harmful.
5. The superiority of salvarsan over mercury and iodid in syphilogenic disease of the nervous system has not yet been proven.
6. Neurologists have in salvarsan a powerful and convenient antisyphilitic remedy when used with proper technic.

After discussing his experience with this remedy in

---

(9) Deutsche med. Wochenschr., No. 35, 1911.

tabes, Nonne makes the statement that salvarsan is generally indicated in the cases where we use mercury, namely in cases which have not been treated for syphilis since the onset of the spinal cord trouble. It can not yet be decided whether salvarsan should be preferred in the rapidly progressive cases with irritative symptoms. It is indicated in cases in which a certain degree of cachexia has set in. Like mercury, it should be used in cases which show a benign course under intermittent treatment. Beginning optic atrophy is no contra-indication providing small doses are given. It is contraindicated in cases in which there are symptoms of lesions of so-called vital centers, particularly of the upper cervical region of the cord and the bulb. That salvarsan accomplishes more in tabes than mercury has not been proven.

As to *paretic dementia* he concludes that salvarsan is no more curative than mercury and the iodids. Marked remissions coming on rapidly after the treatment, have been seen but they are not different from those otherwise seen in this disease. Nonne urges those who reported the results in tabes and paretic dementia quite extensively in the year 1910 to publish the further course of these cases. He considers salvarsan permissible at the incipient stage and as definitely indicated in those cases in which mercury at first acted favorably but is no longer tolerated. In advanced cases which have not been treated or in which mercury has failed, salvarsan is also useless.

As to dosage the question is not settled but Nonne believes that we shall probably decide in favor of small or medium doses.

The nerve recurrences were discussed by Benario who made an extremely interesting compilation of the occurrence of peripheral nerve affections in 194 syphilitics treated with salvarsan and 131 treated with mercury. The following table gives the percentage of some cranial nerve affections under the two kinds of treatment.

	Salvarsan.	Mercury.
Optic Nerve,	29.1%	25.1%
Motor Oculi,	8.6%	11.5%

Trochlear Nerve,	2.3%	.....
Trigeminus,	2.7%	.7%
Abducens,	5.9%	2.3%
Facial,	16.3%	23.0%
Auditory,	35.0%	35.8%

These figures, which in the case of the most important and most frequently affected nerves, namely, the optic and auditory, are nearly equal, strongly support the contention that the nerve recurrences are syphilitic and not toxic manifestations.

Oppenheim's Experience with Salvarsan was thus summarized by himself in the same discussion.

1. Salvarsan may bring about a cure in syphilis of the nervous system. It does not appear to accomplish more than the remedies previously used but has the advantage that a single dose may bring about a cure. It often fails, especially in disease of the brain and cord when mercury and iodid also have failed. It remains for the future to show whether the certainty and permanency of good results are greater with salvarsan than with mercury and iodid. There are cases of cerebral syphilis in which salvarsan may have a harmful effect.

2. In tabes and paretic dementia the positive value of salvarsan treatment is very small. In most cases the patients are uninfluenced, and not rarely the remedy has a harmful effect as the disease immediately or soon after the treatment progresses more rapidly or presents new symptoms. The improvement of the general condition in some cases is counterbalanced by the harmful effect on the same in other cases. With a certain diagnosis of tabes or paretic dementia Oppenheim does not consider salvarsan indicated.

3. What has been already said is also true of other nervous diseases following syphilis, the specific character of which has not yet been positively proven, such as combined degenerations and cases of the type of poliomyelitis. As long as there is a possibility of a genuine syphilitic process the remedy may be given a trial, but for the metasyphilitic degenerations this remedy is probably as useless as the older ones.

4. Oppenheim's experience also shows that severe

symptoms on the part of the central nervous system may appear under the influence of the salvarsan treatment. He is not in a position to decide the question whether we are here dealing with toxic effects of arsenobenzol or with early forms of nerve syphilis. Ehrlich's opinion on these matters is of such heavy weight that others hesitate to advance a personal view.

Oppenheim goes on to say that taking it all together his experience has made him doubtful as to whether our treatment of nervous diseases has been to any material degree advanced by Ehrlich's discovery, as the good features are nullified by the unfavorable effects.

The So-Called Nerve Recurrences After Salvarsan Treatment form the subject of rather sharp exchanges between Ehrlich's fellow-worker Benario[1], and Finger of Vienna. Benario begins by pointing out that Finger's own colleague, working with the same kind of patients in the same hospital, reports a much smaller proportion of nerve recurrences. Thus, in Riehl's clinic there were only 6 clear cases among 435 patients. Only one of these remained permanently deaf on one side. Among the small number of cases treated with mercury alone a case of optic neuritis and one of complete left-sided deafness were observed early during the secondary stage. In Ehrman's clinic, 5 cases of auditory nerve affection and one of papillitis, all with complete recovery, were observed and at the same time there were 2 cases of vestibular nerve affection arising during mercurial treatment. Neisser, who treated 2,000 patients with salvarsan, had 6 nerve recurrences and as many in patients who did not get salvarsan. Since he began to give several small intravenous injections of salvarsan and combined this treatment with mercury, no more nerve recurrences were observed.

Benario sarcastically urges Finger to read carefully the following passage from Neisser's recent monograph on salvarsan treatment: "The existence of peculiar properties injurious to nerves in '606' appeared to me entirely unproven. On the contrary I believe that nearly all cases described as nerve recurrences affecting the cranial nerves should be looked upon as genuine luetic

---

(1) Wien. med. Wochenschr., Feb. 24, 1912.

processes." Klingmüller of Kiel gave over 2,000 intravenous injections to 1,260 patients with 4 nerve recurrences, while he saw 2 cases of optic neuritis, 2 of chorioretinitis and one of papillitis during the secondary stage of untreated cases and 2 cases of facial paralysis and one of abducens paralysis in untreated early lues.

Benario also points to Finger's apparent retreat, as he had made a statement in May, 1911, that "salvarsan undoubtedly possesses neurotropic properties," while now, *post hoc,* he has reached the conclusion that the variations in the symptoms dependent on "intermittent noxes" also may be ascribed to the proteus-like picture of cerebral lues.

Attention is also called to the experiments by Ullmann[2] who failed to find arsenic in the brains of animals injected with salvarsan, and concluded that the chemical examination does not speak in favor of the neurotropic action of salvarsan. As a further proof of Finger's shifting point of view Benario quotes his statement that the nerve recurrences are an "expression of cerebral lues on the basis of syphilitic arteritis; that is, a primary blood-vessel disease with secondary involvement of the cranial nerve." Benario appealed to the famous neuropathologist, Alzheimer, who states: "That salvarsan may cause the Heubner form of arteritis or other important vascular disease which may form the nidus for subsequent localization of spirochets, appears to me as entirely out of the question, at least in experimental arsenical neuritis. I have never seen marked vascular changes but on the other hand, marked nerve degenerations. That salvarsan should cause particular vascular disturbances seems excluded." Finally Benario states that it has been shown that too strongly alkaline solutions of salvarsan may lead to thrombosis.

In his reply Finger advises Benario to read Dössecker's[3] report of the results in Jadassohn's clinic. Here there were 11.1 per cent. of nerve recurrences in patients treated during the first year and observed three months or more after the treatment. Finger also points out that on the same page of Neisser's monograph from

---

(2) Wien. klin. Wochenschr., No. 4, 1912.
(3) Corresp.-Bl. f. Schweizer Aerzte, No. 2, 1912.

which Benario's quotation was taken the following statement appears: "But, nevertheless, it can not be denied that perhaps now more such cases of cranial nerve affections in syphilitics occur through the introduction of salvarsan therapy." [Finger masses together many other quotations but it appears to the unbiased reader that he dodges Benario's direct challenges and fails to make out a good case.—Ed.]

Surgical Treatment. Is Trephining ever Indicated in Brain Syphilis? Horsley's affirmative opinion on this question was stated in the *Practical Medicine Series* 1911, Vol. 10, p. 109. His radical stand that trephining should be resorted to in all cases where a diagnosis of gumma of the brain and its coverings can be made is not generally accepted by neurologists, but on the other hand such an authority as Nonne, who discusses the question in the paper just cited, believes that operation is indicated when epilepsy follows an attack of syphilitic meningitis, and can reasonably be attributed to a meningeal scar. He relates 3 cases of this kind observed during the past year.

Case 1. A middle-aged man, who denied syphilitic infection, had suffered for 8 months from right-sided Jacksonian attacks. There was a transient motor aphasia after each attack. There were no signs of increased brain pressure but the skull was tender over the motor area on the left side. The Wassermann test was positive with the blood, negative with the spinal fluid by the ordinary method, but positive when 8 c. c. of fluid was used. Mercury and potassium iodid did not help and Nonne advised an operation, which was performed and consisted in the removal of an indurated cicatrical area in the dura mater over the face and arm center. Histologic examination showed a gumma. The mercurial treatment was continued and the patient has been well since the operation.

Case 2. A man 30 years old with a history of syphilis had suffered for 2 years from Jacksonian epilepsy limited to the left side of the face and the left arm. There were no pressure symptoms and the specific treatment gave no relief. The Wassermann test behaved as in Case 1 and here also lymphocytosis and positive globulin tests were present. At the operation a walnut-sized, firm, oval gumma was found over the arm and face center. It was removed and the patient has been well since.

Case 3 was also one for operation but the patient died in status epilipticus before it could be performed. A woman 34 years old suffered from severe headache and tenderness behind the right ear. Otologic examination was negative. The 4 laboratory reactions were positive in the same manner as the 2 other cases. The only finding at necropsy was a gumma of the dura of the size of a billiard ball which compressed the temporal lobe.

BRAIN TUMOR

Differential Diagnosis between Hydrocephalus Without Enlargement of the Head, and Brain Tumor, by Means of the X-Ray. It is now universally recognized that the picture of brain tumor may be simulated to perfection by acute or subacute hydrocephalus, generally designated serous meningitis, which condition has been present in a large number of the cases reported as "Pseudo-tumor cerebri." Fortunately, it appears that the *x*-ray may be helpful, as in cases of hydrocephalus there is apt to be deep grooving of the interior of the calvarium by the convolutions of the brain, even in the absence of enlargement of the head. This was found to be the case in a patient examined post mortem by Spiller [4] and reported ten years ago. Recently the same author,[5] in discussing a case presented to the *Philadelphia Neurological Society* for diagnosis by Gordon, pointed out that the *x*-ray pictures showed evidence of such depressions. M. Bertolotti [6] independently has reached similar conclusions. He gives an illustration which shows the impressions in the inner surface of the calvarium, finger-mark like, made by the cerebral convolutions in a case of acute hydrocephalus fatal in 20 days. It might be expected that hypertension and secondary hydrocephalus in cases of brain tumor would cause similar changes in the skull but this does not ap-

---

(4) Amer. Jour. Med. Sciences, July, 1902.
(5) Review of Neurol. and Psychiat., January, 1911.
(6) Revue Neurolog., Jan. 30, 1912.

pear to be the case, as the rise of tension is more gradual.

Another illustrative case is that of a child of 12 years reported by Battiotini and Mattirollo.[7] He was suddenly taken with violent headache, vomiting and stiffness of the neck and presented marked cerebellar ataxia, nystagmus and choked disc. The *x*-ray picture revealed the characteristic impressions in the skull cap. The patient died about three weeks after the onset and the necropsy confirmed the diagnosis of acute hydrocephalus due to ventricular serous meningitis.

The Value of Tuning-Fork Tests in the Early Diagnosis of Brain Tumor was discussed by J. Holinger[8] who related cases showing that there are tumors located in the cerebello-pontine angle which for a long time produce no other symptoms than unilateral impairment of hearing and tinnitus. Among the cases related is that of a man, 42 years old, first seen in March, 1909. He had suffered from occasional headache and gradually lost his hearing in the left ear. He was a little dizzy at times and had some slight left-sided tinnitus. There was no facial paralysis and both ear-drums were found normal. Whisper was heard in the left ear at one inch, and this was not changed by inflation of air through the capsule. Syringing of the left ear with cold boric acid solution did not cause nystagmus or dizziness. There were no other symptoms at the time, but the author felt sufficiently certain of the diagnosis of brain tumor to advise operation. This, however, was refused at the time and performed eleven months later, when a tumor in the cerebello-pontine angle was found. Another patient was a woman, 28 years old, who had been treated for five months for dizziness and noise in the left ear when first seen in February, 1911. She had been the victim of periodic headaches since childhood, but the headache had been worse the last two years. Both tympanic membranes were normal. The patient could hear a whisper in the left ear at 3 cm., and after catheterization at 30 cm. After turning four times to the right, she became very dizzy and had to sit down and there was nystagmus

---

(7) Rivista Critica di Clin. Med., 1911.

(8) Transactions Internat. Otol. Cong., Boston, 1912; only an abstract appears here. The author kindly placed the manuscript at the disposal of the Editor of the Practical Medicine Series.

on looking to the left. On turning three times to the left she became still more dizzy and lost her equilibrium, and there was then nystagmus on looking to the right. She improved for a time on potassium iodid, but a few months later she developed spontaneous nystagmus on looking to the right. Dizziness became more marked and the hearing in the right ear also became impaired. There was no optic neuritis and there was no facial or other paralysis. A diagnosis of tumor in the cerebello-pontine angle was made and confirmed by operation performed in May, 1911, when a walnut-sized tumor was seen and felt in the cerebello-pontine angle, but it could not be removed. Some bone was removed for the decompressive effect, and when last seen in July, 1912, the patient felt well, and has been attending to her household duties. The hearing is a little worse, but she is only occasionally dizzy. The scar of the operation was soft and pulsating.

The following conclusions are arrived at:

1. There are tumors of the cerebello-pontine angle which for many months, even years, cause no other subjective symptoms than one-sided loss of hearing and noise in one ear.

2. The differential diagnosis of these cases from any others suffering from loss of hearing in one ear is based upon the following points:

a. The gradual onset and slowly progressing course.

b. The normal appearance of the drum membrane.

c. A considerable loss of hearing by bone-conduction. This loss is much greater than the loss of hearing by air-conduction. In a fourth case, for example, this symptom was so striking that the patient later on could not hear either an A_1 or an A fork from any part of the cranium, while she still heard whisper, in the good ear at least, at a distance of several meters. Weber-Schwabach's test is strongly negative and heard in the better ear.

3. A careful examination of the vestibular apparatus, caloric tests, static tests, etc., will often help to throw light on the subject. Holinger lays more stress on the finding of any abnormality as such than on the special character of such abnormality. For example,

the caloric test was absent in the second and normal in the fourth case. The opposite was found in the static tests; they were normal in the second, and abnormal in the fourth case. Eye symptoms appeared much later in all four patients. Such symptoms support the diagnosis, but if one waits until all symptoms are well developed, complete recovery by operation will often be impossible, on account of degeneration of important nervous tracts. These patients consult otologists first and the chances for an operation and the final result depend entirely upon their judgment.

Positive Wassermann Reaction in Non-Syphilitic Tumors of the Central Nervous System. Leon Newmark[9] adds two new cases to the list of those which have taught us that there are a few exceptions to the rule that a positive Wassermann test always means syphilis. His first case was one of carcinoma of the breast in a woman 53 years old who developed marked aphasia and later right hemiplegia and choked disc. At the necropsy only the brain and the breast were examined. The cerebrospinal fluid obtained at the necropsy also gave a positive Wassermann reaction. Curiously enough while the tumor of the breast was carcinoma, that in the brain was not a metastatic growth, but a glio-sarcoma, and hence an independent primary tumor.

The second patient was a woman 45 years old who developed symptoms of compression of the cord. A positive Wassermann reaction was obtained both with the blood and the spinal fluid, although the patient had already been treated with mercury, salvarsan and iodid of potassium. As the symptoms grew worse operation was performed and an intradural psammoma removed, following which the patient made great improvement. At the operation, after the patient had been under the anesthetic for an hour, cerebrospinal fluid was removed and gave a negative reaction. Later the blood also gave a negative reaction.

[It must be admitted that these patients were not submitted to sufficient examination to exclude the existence of both syphilis and the non-specific tumors.—ED.]

Multiple Hemangio-Perithelioma of the Brain. W. G.

---

(9) Jour. Amer. Med. Assoc., Jan. 1, 1912.

Spiller [2] reports a unique case, as he has been unable to discover a similar one in the literature. The patient was a man 60 years old who had an attack in February, 1910, when he suddenly became flushed and reeled when he attempted to stand. His speech was a jargon of syllables and numbers so that he could not be understood, and he did not understand what was said to him. His writing was as much disturbed as his speech. He could not walk unassisted. He gradually improved and returned to work, but in January, 1911, he had an attack of convulsions, chiefly right-sided, followed by transient right hemiplegia. There were several tumor-like masses in different parts of the body, the first one to appear being located above the left sterno-clavicular articulation. When examined by Spiller in March, 1911, there was marked paraphasia, word-blindness and word-deafness, and also right lateral homonomous hemianopsia. The tendon reflexes were increased on the right side and there was a right Babinski sign. A few days later he had severe convulsions involving the right side of the face and the right arm, followed by flaccid paralysis of the right side. He died a week later. At the necropsy a large number of hemorrhagic tumors were seen in different parts of the brain, the largest measuring 2 and 3.5 cm. (Plate V, A.) Tumors were also found in the cerebellum and pons. The histologic structure is seen in Plate V, B. One of the tumors of the trunk was examined and found to be an endothelioma. Several of the tumors of the brain were examined and all of them showed the structure of perithelioma.

Malignant Chordoma is a rare form of intracranial tumor which was discussed in the *Practical Medicine Series,* 1910, Vol. 10, p. 85. It arises from the remains of the chorda dorsalis at the junction of the sphenoid bone with the basilar process of the occipital bone and is prone to project into the pharynx and also into the cranial cavity. A new case, the seventh fatal one reported and the largest one on record, is described by Jelliffe and Larkin.[3] The patient was a woman 36 years old, previously in good health, who suddenly developed

---

(2) Jour. Amer. Med. Assoc., Feb. 17, 1912.
(3) Jour. Nerv. and Ment. Disease, January, 1912.

paralysis of the left sixth cranial nerve followed by headache and pain in the area or the left fifth (trigeminal) nerve. Then there developed complete paralysis of all movements of both eyeballs, with blindness, first in the right eye and later in the left eye. The pupils did not react to light or accommodation, and the tendon reflexes in both legs disappeared. There was a gradually increasing somnolence with confusion and dreamy delirium. Later, left facial paralysis appeared, followed by left hemiplegia with hemianesthesia and total paralsis of the fifth cranial nerve. Finally both legs became paralyzed. Marked chemosis of both conjunctivæ developed and two months before death a soft, rounded mass appeared over the sacrum.

The post-mortem examination was confined to the brain but it was also noticed that there was an enlargement extending from the tenth to the twelfth dorsal vertebra in addition to the one over the sacrum. On opening the skull, a large dural tumor was found to extend along the base of the brain from the olfactory bulb to the junction of the pons and medulla. It measured 11 x 6 x 7 cm. The first six pairs of cranial nerves passed directly through the tumor which had grown also through the cribiform plate, filling the nares, and into the sphenoidal sinuses and the maxillary antrum. The orbital plates of the frontal bone, parts of the sphenoid and occipital bones, and the ethmoid, vomer and turbinated bones were destroyed. It is not stated whether or not the tumor projected into the pharyngeal vault.

On account of its gross appearance the tumor was thought to be a myxosarcoma but histologic examination showed it to possess the characteristics of chordoma. The tumors of the dorsal and sacral spine were considered metastases, but sections could not be made.

Tumors of the Fourth Ventricle. K. Bonhoeffer[4] calls attention to the difficulty of differentiating between lesions of the various structures in the subtentorial space, as any lesion in this region causing pressure is prone to produce cerebellar symptoms. Thus, in internal hydrocephalus involving the fourth ventricle, there is apt to appear not only cerebellar ataxia but external

---

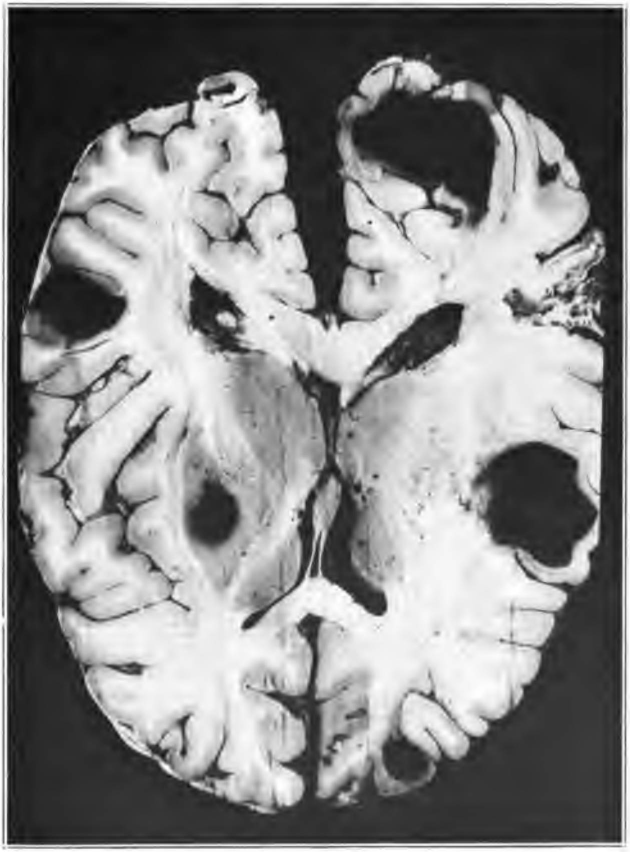
(4) Archiv. f. Psychiat., Vol. 49, p. 1.

rectus palsy, conjugate deviation, disturbance of the corneal reflex, nystagmus and diminution or absence of the knee-reflexes. If the entire clinical history, including the order of appearance of the different symptoms, is known, a correct diagnosis may be made.

Tumors arising from the fourth ventricle itself also offer great difficulty, although many authors, particularly A. Stern, who has thoroughly discussed both tumor and cysticercus of the fourth ventricle, believe that such tumors can be diagnosticated with fair accuracy. Bonhoeffer reports three cases of tumor and two of idiopathic hydrocephalus and is very skeptical about the possibility of clinical diagnosis. He made several punctures of the ventricles both for therapeutic purposes and in the hope that the temporary relief from the pressure caused by hydrocephalus would enable him to recognize the symptoms due to a solid tumor. In both respects he met with disappointment and also saw no prolonged relief from lumbar puncture. In fact he has never seen prolonged relief from lumbar puncture in any case of hydrocephalus and has greater faith in palliative trephining (decompression).

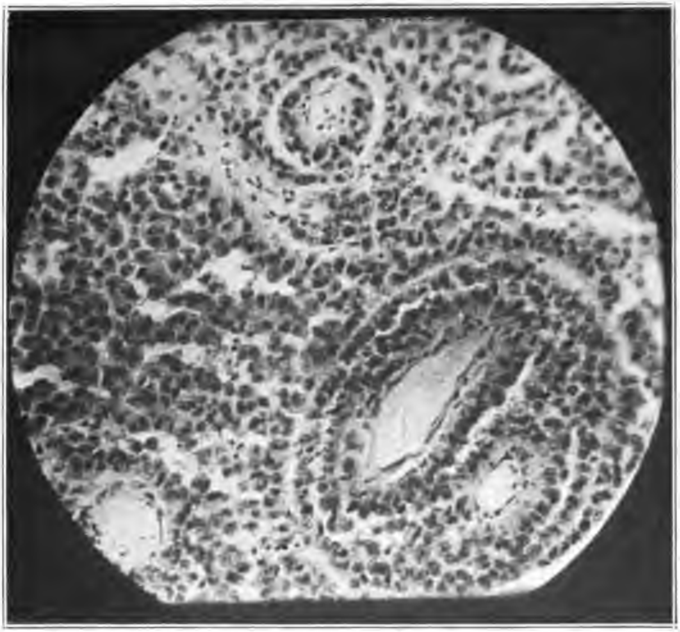
Case 1. A woman 20 years old developed gastric disturbances, with frequent vomiting, in the spring of 1907 and was treated for dilatation of the stomach. At this time she also had severe frontal headache on the right side followed by transient attacks of vertigo and partial fainting. There were occasional attacks of severe pain in the neck; menstruation ceased in August, 1907, and vision began to fail in the right eye in November of the same year and in the left eye four months later. The hearing was good but severe tinnitus developed. On examination in March, 1908, the neck was stiff but not tender. Both discs were choked. There was weakness of the right external rectus and there were nystagmoid jerkings on looking to the left. The right corneal reflex was diminished and there was slight facial paresis on the right side. The tendon reflexes were brisk. Lumbar puncture with removal of 3 c.c. of fluid revealed increase in pressure but no lymphocytosis. Two days later headache and vomiting were severe and the pulse rate was 60. On the next day the patient groaned much

PLATE V, A.



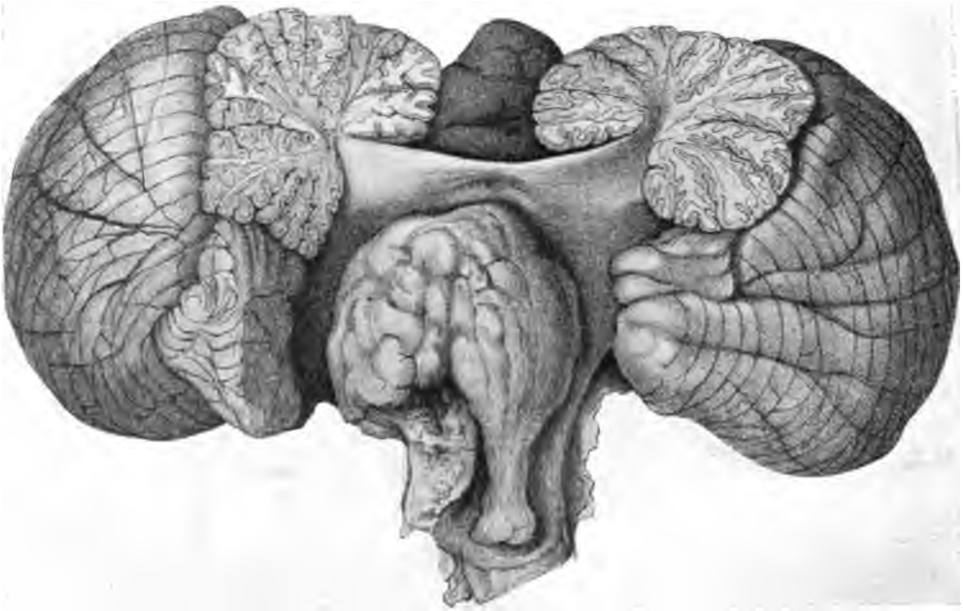
Multiple Hemangio—Perithelioma of the brain.—Gross appearance. —Spiller. (Page 78.)

PLATE V, B.



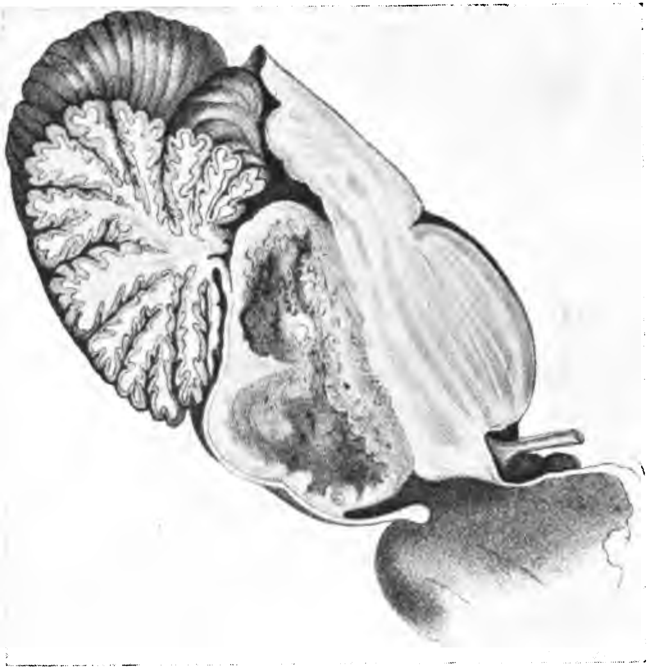
Multiple Hemangio Perithelioma of the brain.—Microscopic appearance.—Spiller. (Page 78.)

PLATE VI.



Glioma of fourth ventricle.—Bonhoeffer. Case 1. (Page 79.)

PLATE VII.



Glioma of fourth ventricle.—Bonhoeffer. Case 2. (Page 79.)

on account of headache and kept the head turned backward and to the left. Both corneal reflexes became weak and there was impairment of the conjugate movement of the eyes to the left. The pulse was rapid and hiccough was present. Both lateral ventricles were punctured and about 50 c.c. of fluid removed. The pain in the head and neck was diminished for a few hours but seven hours after the puncture the patient suddenly died. At the necropsy it was found that the cerebellum was wedged into the foramen magnum in a funnel-shaped manner. There was marked dilatation of the third and lateral ventricles and the fourth ventricle contained a walnut-sized tumor which was found to be a glioma rich in cells. (Plate VI.)

Case 2. A man 20 years old developed severe headaches chiefly in the temples and most severe in the mornings, in February, 1910. There were occasional attacks of pain in the neck, vomiting, yawning and eructations. Later there was also severe pain in the shoulders. When he was examined two months later the head was held stiffly to the left and backwards, the points of exit of the occipital nerves were tender, but the head was not tender on percussion. Optic neuritis with secondary atrophy was present though vision was good. The ocular movements were limited on both sides, more so to the left, and the left corneal reflex was diminished. Smell, taste and hearing, as well as the tendon and cutaneous reflexes, were normal. There was considerable ataxia with a tendency to fall to the right. Lumbar puncture revealed a moderate increase of lymphocytes and a strongly positive globulin reaction. Two weeks later the patient suddenly became unconscious and cyanotic. Both lateral ventricles were punctured without result. The left side of the cerebellum was punctured and no change found in the tissue obtained. The patient improved very rapidly, but a day later a second and similar attack occurred, followed in three hours by death. In this case also the cerebellum was found incarcerated in the foramen magnum and the fourth ventricle was filled by a gliomatous tumor. (Plate VII.) The aqueduct of Sylvius was closed and the central canal of the cervical portion of the spinal cord was markedly dilated but there was no degeneration of the white tracts of the cord on histologic examination.

Case 3. A boy 11 years old first complained of vomiting and headaches; he soon became absent-minded at school and began to stagger, with a tendency to fall to the left. There was a Babinski sign on the left side, the left corneal reflex was diminished and the lower facial nerve appeared slightly paretic at times. Bilateral choked disc was present. Lumbar puncture revealed a slight lymphocytosis and negative globulin reaction. Ventricular puncture with the removal of 30 c.c. of fluid resulted in relief of headache for a few days but the choked disc and other objective findings remained unchanged. Three weeks later he had two attacks of unconsciousness with rigidity and upward rotation of the eyes. Another ventricular puncture was made followed by another attack of rigidity and the appearance of the Babinski sign, ankle and patellar clonus on both sides. The next day the patient was free from headache and in good spirits. Two weeks later he had another attack of unconsciousness. Forty c.c. of fluid were removed by ventricular puncture. The knee- and ankle-jerks now disappeared. During the following four weeks he had considerable headache, very variable pulse and occasional attacks of vomiting. Two more ventricular punctures were made and the patient died five days after the last one. The fourth ventricle was the seat of a large glioma and all the ventricles were greatly dilated.

In commenting on these cases the author points out the onset with headache and vomiting in all of them and that the headache in Case 1 was chiefly frontal, in Case 2 chiefly temporal. As two of these patients kept the head backward he does not agree with Stern's dictum that the forward position of the head is characteristic of tumors of the fourth ventricle. The presence of choked disc in all of the cases contradicts the statement of Bruns that choked disc is absent in fourth ventricle tumors. A tendency to sudden attacks of various kinds, such as vomiting, vertigo, tonic spasms, coma and cyanosis, etc., existed in all of the cases. They all showed cerebellar ataxia but there were no local symptoms referable to the medulla oblongata, and the tumors had not invaded this structure. The presence of paresis of the conjugate eye movements in all three cases is in contrast with the experience of Uhthoff who found this disturbance to be rare in tumors of the fourth ventricle. Its occurrence in the three present cases is explained by the upward growth of the tumor into the aqueduct of Sylvius, which existed in all of them.

For purposes of comparison the author describes two cases of idiopathic hydrocephalus the cause of which remained unexplained by the post-mortem findings.

Case 4. A woman 21 years old was taken ill with chilly sensations, dizziness, vomiting and headache followed by two attacks of convulsions with unconsciousness. A month later blindness set in, the eye-grounds showing choked disc with secondary atrophy. There was weakness of the conjugate movements of the eyes to the right and of the right external rectus. The corneal reflexes were diminished. The right arm was weaker than the left. Babinski's sign was present on both sides. The gait was unsteady. When standing with the feet together there was a tendency to fall backward. Later ventricular puncture was made which was followed by infection and fatal meningitis. There was no tumor, but marked hydrocephalus with dilatation of all the ventricles.

Case 5 was that of an 8-year-old boy suddenly taken with nausea and vertigo and stiffness of the neck and back and at first treated for rheumatic torticollis. Later the gait became unsteady, and blindness set in six weeks after the onset. Headache and vomiting were not noted. There was bilateral choked disc with secondary atrophy, and the right external rectus was weak. The corneal reflexes were present. The cerebrospinal fluid obtained by lumbar puncture was under slightly increased pressure and showed slight lymphocytosis and increase in proteids. An attack of sore-throat was followed by severe occipital pain. This pain increased and was not relieved by two lumbar punctures. The choked disc also increased and the patient died after a few days. The necropsy revealed marked internal hydrocephalus.

Both of these cases came under the observation of the author after the onset of blindness and he emphasizes the importance of early trephining in such cases, taking the condition of the optic disc as an indication rather than the vision.

According to Gonzalo R. Lafora[5] most cases of tumor of the fourth ventricle have occurred in children. Glioma is the most common form of tumor. The clinical manifestations are variable; sometimes they are bulbo-pontine, sometimes cerebellar in character. To the first belong facial paralysis, glycosuria, dysphagia, dysarthria, albuminuria, Cheyne-Stokes respiration, and paralysis of the palate and of the tongue; while to the second belong cerebellar ataxia, vertigo, adiadochokinesia, a tendency to fall backward or to make rotary movements with the whole body, and priapism. Death is sometimes sudden. All these different symptoms of one character do not appear constantly in any case. A symptom which occurs very frequently is periodic dizziness; in the cases of echinococcus cyst this is the most prominent symptom, and it appears not only periodically, but also at the time of performing sudden rotatory movements, especially in cases of loose cysts (Brun's symptom). There have been described, however, some cases of tumors of the fourth ventricle by Douty, Zenker, Verron, and, latterly, two by Besta, in which mental symptoms were prominent. In the case reported here they were more marked, while focal symptoms were almost absent. In the first case of Besta's (a woman of 54 years), attacks, with loss of consciousness and falling (duration from ten to thirty minutes), without aura or convulsions, were observed every two or three days. Similar phenomena appear to have occurred in other reported cases as well as in the one described here. In many instances trauma seems to have had a very important influence on the causation of the disease. This factor is present in the case here reported, although it is not possible to determine how much influence it had, because of the uncertain epochs of the origin of the tumor and because of the early production of the trauma.

The histologic nature of these tumors is often glio-

---

(5) N. Y. Med. Jour., Nov. 18, 1911.

matous. They arise from the subependymal neuroglia of the oblongata, pons, cerebellum, or velum medullaris. Sometimes the ependymal epithelium which covers the tumor, develops and gives rise to an ependymoglioma. The tissue of the tumor remains always well differentiated from the surface of the oblongata; the oblongata seems to have a marked resistance to the penetration of the tumor, and the ganglion cells of the different nuclei appear well preserved. In the mass of these gliomata there are often found small cavities, which sometimes are covered with a layer of cylindrical epithelium—probably the remnant of the ventricular epithelium. It is, however, well worth while to remember that such cavities have been often described in all kinds of gliomata and that Stroebe has even described gliomata of the brain cortex in which small cysts, covered with a layer of cylindrical or cuboid epithelium cells, were found. These and other facts have induced some authors to believe a disturbance in development to be the origin of the gliomata.

The author's case is unusual in that the patient was 63 years old. The following were the chief points in the case: A cranial injury was sustained seventeen years previous to his death. Following the injury there was observed a change in the conduct of the patient, especially manifested in the form of attacks of depression and excitation, marked religious tendency and frequent conflicts with others. Two years ago the patient showed a self-appreciation (insight) of his mental trouble. There was no history of syphilis or of alcoholism. When brought to the hospital, in consequence of a recent conflict on the street, the patient appeared to be very excited during the eight days he remained in the hospital. There was also great motor activity, but without distractibility or tendency to the rapid associations observed in the manic, type of cases. The continuous bath did not produce any beneficial effect. Later, there were rapid respiration and pulse with slight cyanosis of the hands, without evidence of pulmonary or cardiac involvement, and these conditions continued until the patient died.

The findings corresponding with this symptomatology were: A tumor in the fourth ventricle (ependymoglioma) of the size of a hazel-nut, which did not penetrate the tissue of the medulla, and a small angioma in the lemniscus medialis in the pons. Besides this, a number of miliary plaques were found in the cortex of the brain, which very likely caused the special mental trouble developed by the patient.

The vasomotor perturbations produced by both tumors, compressing the oblongata and pons, seem to have contributed to that form of psychosis in this case. It is not possible to say how much influence the trauma, seventeen years previous to death, had on the production of both tumors.

The Value of Decompressive Operation on the Head. C. H. Frazier[6] gives a summary of his extensive experience with this operation and arrives at definite conclusions concerning its limitation and indications. He is convinced that it has passed successfully through the experimental stage and that it will continue to be found a useful procedure in a great many cases of brain tumor. On the other hand he has little faith in its usefulness in cases of recent cerebral trauma and in epilepsy. As to the former, he says: "The failure of cerebral decompression to relieve such ill effects of intracranial pressure as are the direct result of cerebral contusion are not difficult to explain. In addition to the more or less diffuse lacerations and hemorrhages, minute as they may be, perhaps the most serious phenomenon of cerebral contusion is the rapidly developing cerebral edema. No matter which theory is accepted as to the development of the edema, we know it to be a progressive process, often without limitations, involving the brain structure as a whole. The course and the extent of the process bears a direct relation to the violence of the fracturing force, and the mere establishment of an opening for the relief of tension cannot exert any inhibiting effect upon the process. While the degree of intracranial tension may be intense, death is not attributable to this alone, harmful as it may be, but rather to direct injury and involvement of important vital centers, and perhaps also to the circulatory disturbances caused by the more or

---

(6) Univ. of Penn. Med. Bull., February, 1912.

less intense edema. The conditions prevailing in cerebral trauma and cerebral tumor have this essential difference that, on one hand, we have a lesion rapidly becoming diffuse and unmanageable in which the vital centers become directly involved; on the other, a lesion involving a limited area, of slow development, involving only indirectly the important vital centers. Decompression in the one case is usually futile; in the other, frequently alleviating. At one time I was inclined to believe that by proper differentiation one might select a group of cases midway between the mildest and the most severe in which the establishment of an opening in the skull and dura might be justifiable, but I am now almost convinced that the fatal course of none can be averted by decompression."

As to epilepsy, he states that decompression was first employed in this disease by Kocher on the theory that immediately prior to the attacks there was a condition of hypertension. Frazier's experience, based on operations on 53 cases, has led him to conclude that decompression is of no value [See "Epilepsy" in this volume for full account of Frazier's views on the surgical treatment of that disease.—Ed.]

As to *cerebral tumor,* he rightly states that so much has been written of late upon the subject of decompression that the very much more important consideration, the radical removal of the growth, has been overshadowed. As Sir Victor Horsley says, the palliative operation should be the exception and free extirpation the rule. In cases which apparently were permanently relieved by decompression, some doubt as to the correctness of the diagnosis exists unless the patient finally comes to autopsy. Internal hydrocephalus, meningitis serosa and other conditions may, for a time, simulate tumor almost to perfection.

"While decompression itself is not difficult of execution, it is not always easy to determine when to decompress, or whether, prior to decompression, one should search for the tumor. As to the latter, in earlier communications I have maintained that exploration should precede decompression whenever there is the slightest suggestion as to the seat of the tumor; that only by frequent resort to exploration will the percentage of operable tumors be increased. In principle I still adhere to this point of view, but I find in looking over my records that while exploration preceded decompression in 66 per cent., or about two-thirds of my cases, in only 17 per cent. of them was the tumor found. Even though the percentage be small we should continue to search for the tumor, since there is little if any added risk in the combined operation, and occasionally one will find a tumor which can be extirpated.''

In critical cases with excessive tension both clinical and experimental evidence has taught him that no more immediate benefit is derived from a bilateral than from a unilateral opening. He has found that the usual subtemporal operation relieves tension as effectually as one in the subtentorial or suboccipital region but maintains that if there is any evidence, be it ever so slight, for believing the tumor to be subtentorial a suboccipital craniectomy should be performed and the cerebellar fossa explored.

As to selection of cases for operation he says: ''Should we invariably resort to decompression in every case of inoperable or unlocalized brain tumor? In the earliest days of the operation an affirmative answer might have been given but experience has taught us that, under certain circumstances, the operation has little or no effect upon the fatal tendency of the lesion. The operation itself is designed to relieve tension, but increased tension is not invariably associated with the presence of a tumor. In tumors of the infiltrating type, particularly the gliomata, the brain is destroyed as the tumor advances, and there is often no material increase in the total amount of tissue within the cranium, even though the tumor be of large dimensions. We are in the habit of attributing all the symptoms which are not focal to this increased tension, but as our observations multiply, we find there are certain exceptions. While headache and vomiting may be due solely to excessive intracranial pressure, there must be some other factor than mechanical pressure to account for papilledema, and the same is true of the attacks of coma, often periodic, but usually exhibited in the latter stage of the disease.''

As stupor had been a prominent feature in his only five fatal cases of decompression he considers this almost an absolute contra-indication.

"As to the influence of decompression upon headache and vomiting, one or the other, or both, are relieved in about two of every three cases, and in subtentorial lesions the vertigo is almost invariably relieved, at least to some extent. However, there is nothing which so disturbs the patient as threatened loss of vision. Frequently headache, nausea, vomiting and any other harmful effect of tumor will be tolerated with extraordinary patience and endurance, but once the sight begins to fail and the gravity of the situation realized, the patient grasps at any proposal which will give him a ray of hope. Unfortunately, I am ashamed to say, in one out of every three cases the condition of the disc was such that restoration of vision was out of the question; a truly pitiable commentary, where carelessness, procrastination or misplaced faith in the effect of mercury or the mysteries of osteopathy, perhaps only one, perhaps all successively, combine to rob the patient of the one chance of restored vision. I have been fortunate in having in almost all my cases the advice of George E. de Schweinitz, who has devoted much time and labor in studying the expression of papilledema in brain tumor, especially with relation to decompression, and in presenting to you the effect of decompression upon papilledema I can do no better than quote from his most recent contribution.

"The most satisfactory treatment for the purpose of preserving vision in any case of choked disc or papilledema not due to a toxic process or constitutional disease ('infectious optic neuritis'), but depending upon increased intracranial tension, is decompressive trephining.

"This operation should be performed early, and if it can be done during the first, second, or even third stage of papilledema the prognosis as to sight is favorable.

"If for any reason the operation is postponed until the development of the fourth and fifth stages of papilledema, already associated with marked depreciation of vision, the prognosis as to sight is most unfavorable, but even under these circumstances the operation should be performed, because it sometimes preserves such vision as still remains.

"In non-syphilitic cases, time devoted to the administration of iodids and mercurials is time wasted, but after the operation their exhibition appears to exert a beneficial influence.

"Patients afflicted with papilledema depending upon increased intracranial tension should have the case fairly stated to them, and the operation should be urged in spite of the occasional unfavorable results, because in its absence ultimate blindness is almost sure to result."

In conclusion the author makes the following summary:

1. The fatal tendencies of cerebral contusion with or without basal fracture are nowise averted by decompression.

2. Cerebral decompression, in the experience of the author, has no more influence in any form of epilepsy than that of operations *per se.*

3. In decompression without exploration the element of uncertainty as to the diagnosis must be given due allowance when compiling statistics. Internal hydrocephalus, meningitis serosa and other conditions may so simulate tumor that the real nature of the affection is not determined unless revealed at necropsy.

4. Whenever a general anesthetic is contra-indicated, temporal decompression should be performed under regional anesthesia by an alcoholic injection into the third division of the trigeminal nerve.

5. In critical cases with excessive tension, experimental and clinical evidence indicates that no more immediate benefit is derived from a bilateral than a unilateral opening; and, secondly, that a contralateral opening gives no appreciable relief to the state of tension on the opposite side.

6. From observations in the experimental laboratory it has been found that a pretentorial or temporal opening as effectually relieves the tension as one in the subtentorial or suboccipital region.

7. In the selection of cases for decompression, exclude those with coma or stupor, either periodic or continuous, as they are both indicative of the terminal stage of the

disease and are not influenced as are other symptoms by an artificial opening.

8. Simple decompression without exploration in properly selected cases is so free from risk that the danger of operation *per se* need not be reckoned with when dealing with a condition evidently fatal and often calling for immediate relief.

9. The expectation of life varies according to the seat and character of the tumor. In the author's service, the survivals include periods of three, four and six years.

10. After decompression, headache, vomiting and vertigo are relieved altogether or in large measure in most cases.

11. In patients afflicted with papilledema, ultimate blindness is almost always sure to result unless in the earlier stages the brain is decompressed. When performed early the prognosis as to sight is most favorable.

12. Decompression properly executed and judiciously employed has survived the period of probation and has more than substantiated the forecast of earlier writers.

THE BASAL GANGLIA.

Progressive Lenticular Degeneration: A Familial Nervous Disease Associated with Cirrhosis of the Liver. This is the title of one of the most noteworthy recent contributions to neurology, made by one of the ablest of the younger generation of investigators connected with the principal seat of scientific neurology in England, namely, the *National Hospital* at Queen's Square, London, the institution where Hughlings Jackson, Sir William Gowers, Sir Victor Horsley, Bastian and Beevor have done their principal work. The author is S. A. K. Wilson,[7] who has correlated four cases of his own, with two reported by Gowers in 1888 as "tetanoid chorea associated with cirrhosis of the liver;" one by Ormerod in 1890 as "case of cirrhosis of the liver in a boy, with obscure and fatal nervous symptoms;" and three (two brothers and a sister) by Homén, of Helsingfors, in 1890, as "a peculiar disease occurring in three mem-

---

(7) Brain, 1912, Vol. 34, pp. 295-509.

bers of a family in the form of a progressive dementia, probably lues hereditaria tarda."

Definition: Progressive lenticular degeneration may be defined as a disease which occurs in young people, which is often familial but not congenital or hereditary; it is essentially and chiefly a disease of the extrapyramidal motor system, and is characterized by involuntary movements, usually of the nature of tremor, dysarthria, dysphagia, muscular weakness, spasticity, and contractures with progressive emaciation; with these may be associated emotionalism and certain symptoms of a mental nature. It is progressive, and, after a longer or shorter period, fatal. Pathologically it is characterized predominantly by bilateral degeneration of the lenticular nucleus, and in addition cirrhosis of the liver is constantly found, the latter morbid condition rarely, if ever, giving rise to symptoms during the life of the patient.

Clinical Conclusions: 1. Progressive lenticular degeneration is a disease of the motor nervous system, occurring in young people and is very often familial. It is not congenital or hereditary.

2. It is progressive and fatal within a varying period; acute cases may last only a few months; one chronic case as a maximum continued for seven years; the average duration of chronic cases is four years.

3. It is characterized by a definite symptom-complex, whose chief features are: Generalized tremor, dysarthria and dysphagia, muscular rigidity and hypertonicity, emaciation, spasmodic contractions, contractures, emotionalism. There are also certain mental symptoms, either transient and such as one sees in a toxic psychosis, but not severe, or more chronic, consisting in a general restriction of the mental horizon, and a certain facility or docility without delusions or hallucinations, and not necessarily as progressive as the somatic symptoms. The mental symptoms may be very slight and are sometimes absent.

4. In pure cases the affection constitutes an extrapyramidal motor disease, for the reflexes are normal from the point of view of the function of the pyramidal tracts.

5. The neurologic symptoms constitute a syndrome of the corpus striatum, which has not hitherto been differentiated in this disease.

6. In some ways the disease bears a resemblance to paralysis agitans, and throws light on the problem of that affection.

7. Although cirrhosis of the liver is constantly found in this affection, and is an essential feature of it, there are no signs of liver disease during life.

Pathologic Conclusions: 1. The chief pathologic feature of the disease is bilateral, symmetrical degeneration of the putamen and globus pallidus, in particular the former.

2. This degeneration is the sequel to the selective operation of some morbid agent on the cells and fibers of the putamen and lenticular nucleus generally. The caudate nucleus is often somewhat atrophic, but never to the same extent, while other large collections of gray matter in the immediate neighborhood of the lenticular nucleus, *e. g.,* the optic thalamus, which has partially the same blood-supply, are not affected at all in a pure case unless it be indirectly, and to a very slight extent.

3. The morbid agent is probably of the nature of a toxin.

4. A constant, essential, and in all probability primary feature of the pathology of the disease is cirrhosis of the liver, not syphilitic or alcoholic; it is multilobular or mixed in type, always pronounced, but presenting a varying pathologic picture of necrosis, fatty degeneration and regeneration.

5. It is probable that the toxin is associated with the hepatic cirrhosis, and may be generated in connection therewith. An important analogy may be drawn from the occurrence of *Kernikterus* in certain cases of familial icterus gravis neonatorum, where, in spite of the universal bile-staining of the tissues of the body, certain collections only of gray matter in the brain show a marked affinity for the circulating poison, while others do not. The parts that are stained deeply are in particular the nucleus lenticularis and the corpus Luysii (among others), while the optic thalamus, for instance, is scarcely stained at all.

6. The pyramidal tracts are intact from Betz-cells to muscles in a pure case; occasionally certain secondary changes occur of limited significance.

7. Certain secondary degenerations in the subthalamic region, of physiologic importance, follow on the lenticular disease.

Physiologic Conclusions. 1. The corpus striatum exercises a steadying effect on the action of the corticospinal system.

2. This is effected either by the lenticulo-rubrospinal system, or more indirectly *via* the optic thalamus and its cortical connections.

3. When this influence is impaired pyramidal function is affected in its turn, and is seen in hypertonicity or rigidity, as well as in tremor on voluntary movement.

4. There is not, however, any paralysis in the strict sense.

5. The direct connection of the corpus striatum with the cortex is minimal.

6. There is no necessity to postulate articulatory "centers" in the putamen or globus pallidus.

7. Dysarthria may result without any pyramidal involvement of genu fibers, and with intact cranial nuclei, from hypertonicity of the musculature concerned.

8. Tremor is due more particularly to failure of function of the lenticulo-rubrospinal system.

9. Hypertonicity or rigidity of the musculature, due to defect of the "inhibitory" action of the corpus striatum, is possibly associated with impairment of impulses from that body *via* the optic thalamus to the cerebral cortex.

Illustrative Case. The author's Case 1 is summarized by him as follows:

A young woman, aged 21 whose family history is negative, and who has never suffered from any particular illness, who has always been intelligent and physically active, suffers from an attack of jaundice of about five weeks' duration, the exact details of which are not forthcoming, and at intervals thereafter, for about two or three years, has occasional swelling of the ankles, without being in any way incapacitated thereby. Four years after the icterus, at the age of 25, she notices that her right hand shakes a little as she is writing, and her articulation becomes a little slurring. In the course of a few months her friends notice a considerable alteration in her general condition; she is restless, unable to settle to anything, easily provoked to laughter, constantly smiling and unnaturally cheerful. At the same time the tremor spreads to both arms and hands, her writing deteriorates, her articulation is definitely impaired, and she has some trouble in swallowing.

At the age of 26 she is examined by a neurologist, who can find no signs of organic disease of the nervous system, notes that there is no nystagmus, and obtains a double flexor response. Nevertheless, the condition is steadily progressive; the tremors are accentuated, the dysarthria and dysphagia increase, a generalized stiffness of the musculature reveals itself, and the fingers begin to assume certain attitudes of contracture. Her mental condition is one of facility; she is easily amused, and constantly laughing; her mouth is open and saliva occasionally escapes involuntarily. In spite of the vacant expression on her face her memory and perception are quite good; she has neither delusions nor hallucinations; she is very observant and often makes apposite remarks about those with whom she is associated. The symptoms are characterized by a curious variability; articulation and the power of swallowing seem sometimes to improve, and there is no true paralysis, in the sense that all voluntary movements can be carried out, though slowly.

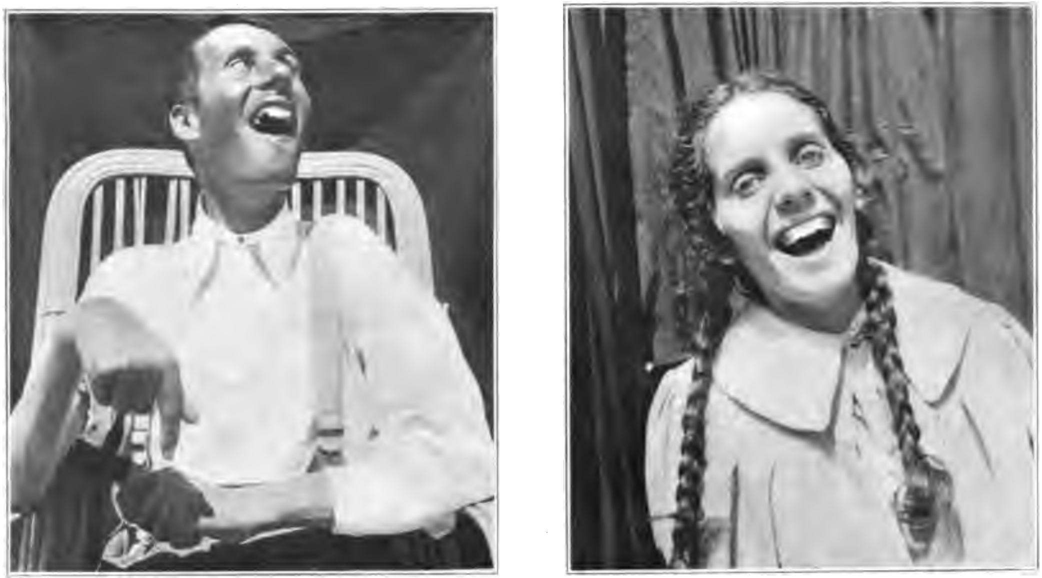
During the next two years the disease slowly progresses. While for a long time bodily nutrition is well maintained, the other symptoms increase in severity. The features are fixed in a perpetual smile; the mouth is wide open and the sialorrhea is more marked; the patient is anarthric and dysphagic; yet with voluntary effort the mouth can be slowly closed, the tongue slowly protruded, while the palate rises on the attempt to articulate; the pupils react briskly to light, ocular movements are free, and the optic discs are normal. The muscles become more and more rigid, the arms, and to a less extent the legs, fixed in attitudes of contracture, which can be to some extent, but not entirely, overcome by passive movement; as a result the patient has become peculiarly helpless; but again on volitional effort a considerable range of movement is still possible. The tremor is absolutely constant, often wide in range and severe in degree; it affects all muscular groups in the limbs, especially the distal groups, but includes also the lower jaw, head, neck and trunk. No sensory change can be detected; the deep reflexes are present without being exaggerated, there is no ankle-clonus; the abdominal reflexes are diminished, and the flexor response on the left side has changed to extensor, the other remaining as before. Defect of control over the sphincters, probably of central origin, appears.

Eventually emaciation sets in; the patient is reduced to a profound degree of helplessness, is in a state of contracture, speechless, incontinent; nevertheless, her mental condition remains clear, she understands everything and endeavors to express her wants. An acute attack of hematemesis ushers in the end, and she dies at the age of 29, after an illness of rather more than four years. With the exception of the initial symptoms nine years previously, there have never been either symptoms or signs referable to disease of the liver, but this is suspected during life and confirmed at the autopsy.

A summary of the pathologic findings is as follows: The brain is of good size, shape and weight. The cerebral gyri are not atrophic, are of normal convolutional pattern, and present no obvious morbid appearance. The membranes are to all intents and purposes normal. The cerebral blood-vessels are not thickened or occluded, and show no patches of disease in their walls.

On Marie's *coupe d' élection,* the eye is at once caught by a complete bilateral and symmetrical destruction of the lenticular nucleus. (Plate VIII, A.) In place of the latter, on either side, and more especially in place of its outer and middle zones, is an elongated cavity, measuring 2½ cm. long by 1½ cm. wide, with dark-colored crumbling walls, extending from the anterior, to the posterior limit of the putamen, and from its extreme lower extremity almost to its upper limit. (Plate VIII, B.) Only a small piece of the inner

PLATE VIII.

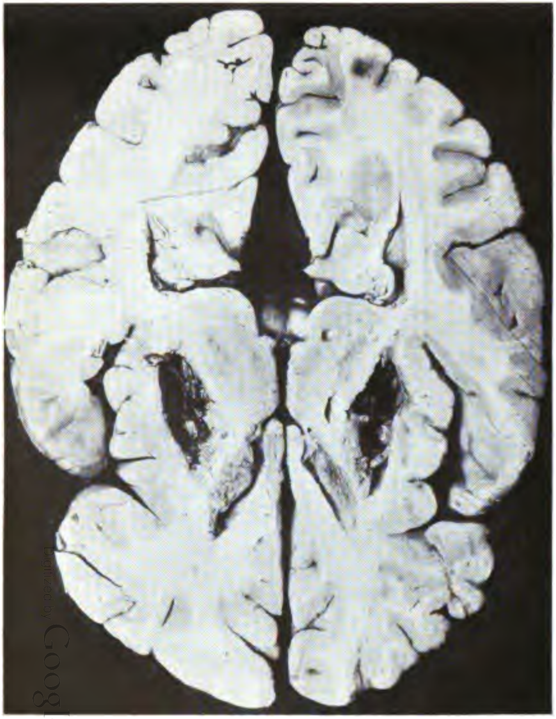


A

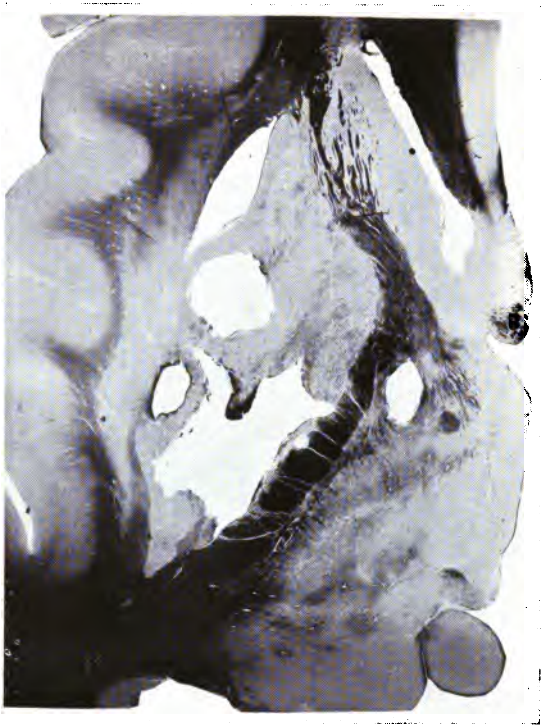
B

Appearance of patients with progressive lenticular degeneration.—Wilson. (Page 91.)

PLATE IX.

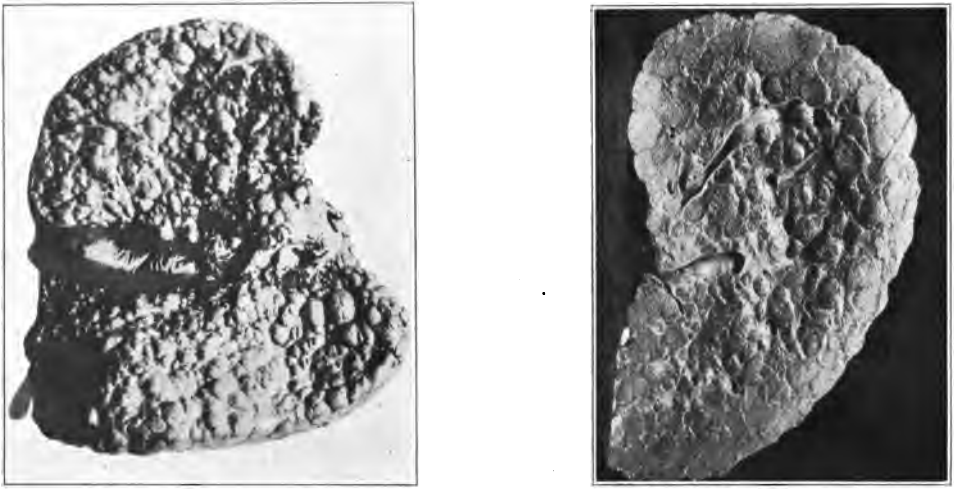


gitudinal section of brain in case of progressive
r degeneration.—Wilson. (Page 91.)



Destruction of the lenticular nucleus in progressive lenticular degeneration.—Wilson. (Page 91.)

PLATE X.



A.

B.

The liver in progressive lenticular degeneration. A. Under surface. B. Cut section.—Wilson. (Page 91.)

zone of the globus pallidus remains. Compared with this utter degeneration of the lenticular nucleus, the optic thalamus and the caudate nucleus are well preserved, except that in the former, on the left side, there is a small punched-out hole towards its posterior part, and the latter is on both sides a little shrunken, and less full and rounded than in a normal brain. The degenerated area extends close up to the internal capsule on both sides; on the right this seems quite intact throughout, although it is found to be somewhat undermined; on the left there is a small prolongation of the cavity across the genu of the capsule, the fibers of which are separated rather than destroyed. Microscopically, however, there is some descending degeneration in the genu fibers.

The external capsule is thinned on both sides, and degenerated in its middle third; the claustrum is very slightly invaded on the left side; the cortex of the island of Reil is well preserved on both sides, although microscopically there is some degeneration of the subcortical fibers of its convolutions towards its posterior part.

The white matter of the cerebral hemispheres is normal. The cortex is practically normal; in particular the origin of the pyramidal tracts in the Betz-cells of the motor area is carefully examined, and the great majority of the latter are found to present no morbid appearance, although some show chronic degenerative changes.

With the exception of the slight descending degeneration in some capsular genu fibers on the left side, the pyramidal tracts stain normally throughout, and, followed from the motor cortex through the capsule, crus, pons, medulla and cord, are perfectly normal.

On the other hand, the extrapyramidal system from the lenticular nucleus, *viz,* the ansa lenticularis, to the red nucleus, is degenerated on both sides; the lenticular bundle of Forel is partly degenerated, as are a large number of the strio-Luysian fibers on both sides. The corpus Luysii is smaller than normal. The cells of the red nucleus do not appear changed, nor does the nucleus seem altered in its fiber-content. The strio-thalamic fibers are degenerated.

The external medullary lamina of the optic thalamus, the *zone grillagée,* and the outer part of the thalamus generally, are to a certain extent altered.

The pons and medulla, and the cerebellum, are not the seat of any change that can be recognized microscopically. The nuclei of the cranial nerves are unchanged, except for slight alterations of secondary significance.

The spinal cord (cells and fibers) is normal, except for certain slight alterations, also of secondary significance. The same is true of the muscles.

The liver is in an advanced state of cirrhosis; the type is mainly multilobular, but some monolobular cirrhosis is found. In the cirrhotic tissue abundant bile-ducts ramify. The hepatic cells are in many instances normal, others are necrosed, many show fatty infiltration and degeneration, others are actively regenerating. The spleen is not enlarged. The thyroid shows interstitial changes. There are evidences in all the organs examined, particularly in the liver and nervous system, of a terminal bacterial infection.

A New Symptom-Complex Resembling Pseudo-Sclerosis, is described by Bruno Fleischer,[8] who reports three cases presenting the following principal features: Tremor, psychic disturbances, brown pigmentation of certain tissues, particularly the periphery of the cornea, and cirrhosis of the liver. Some cases of this kind showing pigmentation of the cornea have previously been reported, among them being two cases by the present author. All of them showed symptoms of a severe nervous disease which was interpreted as multiple sclerosis, but the author three years ago expressed doubt on this point and suggested that we were dealing with a new symptom-complex. Since then he has had opportunity to examine post-mortem one of the first cases reported.

Case 1. A farmer, born in 1878, was first admitted to the clinic in 1901 with the history that between the ages of 12 and 19 years he had had four attacks of severe pains in the extremities, that the legs had been weak since the age of 15 years, and that at 20 years speech began to be affected and a coarse tremor to develop which gradually increased in severity. The

---

(8) Deutsche Zeitschr. f. Nervenheilk., Vol. 44, p. 179.

tendon reflexes were increased and he had ankle-clonus, but no sensory or ocular findings at that time, and a diagnosis of multiple sclerosis was made.

In 1901 a peculiar condition of the cornea was discovered and described by Kayser. In the deep layers at the periphery there was a greenish discoloration and with a magnifying glass minute yellowish granules could be seen. In 1902 some unsteadiness was noted and in 1904 the patient complained of not seeing as well as formerly at night, but central vision was normal. He gradually grew worse and in 1907 the tremor had grown so severe that he could not feed himself. The abdominal reflexes, which had been normal in 1902, were absent in 1907. The cerebrospinal fluid was normal. The brownish-green ring in the cornea gradually grew broader. The tremor began to involve head, arms and legs. In 1909 an albuminous substance and sugar were found in the urine but the sugar elimination was not increased by feeding glucose to the patient. The liver was enlarged, but the heart, arteries and lungs showed no changes. The patient became demented, apathetic, at times stuporous, would scold in an explosive manner and express delusions of persecution. He became emaciated and finally died of erysipelas. The clinical diagnosis was multiple sclerosis.

The necropsy findings were very interesting. The kidneys showed brownish pigmentation in the pyramids. The liver was small, of the hob-nail type, with brownish and yellowish pigmentation. The parenchyma of the testicle showed a grayish-brown pigmentation. The pia between the cerebral hemispheres also showed an area of grayish-brown pigmentation, otherwise there was no gross change in the central nervous system. There was also some pigmentation of the skin. Microscopic examination of the brain and cord showed no changes of any kind. The cornea showed pigmentation of Descemet's membrane, and there was also pigmentation of the choroid. There was a little pigmentation of the muscles attached to the eyeball.

Case 2. A man 29 years old developed some gastric disturbance which caused him to change his occupation from merchant to photographer. Two years later he lost his fortune quite suddenly and then developed nervous symptoms, chiefly tremor of the hands. The latter became so severe that within two months he became unable to write and in three months unable to feed himself. Some months later his legs began to tremble, he developed states of mental excitement, would break furniture and threaten to kill his wife. He had to be admitted to an institution for the insane. Aside from the marked coarse tremor which was increased on voluntary movements he showed marked ataxia of the upper extremities, but not of the lower, and increased tendon reflexes, but the plantar and abdominal reflexes were normal. The urine at one time gave a positive sugar reaction. Examination of the eyes showed the same type of pigmentation of the cornea as was shown in Case 1. The diagnosis at that time was hebephrenia and pseudo-sclerosis.

There was no marked change in his mental condition from this time until his sudden death three years later. The chief post-mortem findings were cirrhosis of the liver of the hob-nail type, there were interstitial changes in the kidneys, some degeneration of the heart muscle, atheroma of the aorta and coronary arteries, hypertrophy of the left ventricle, chronic splenic enlargement, chronic gastric catarrh and copious hemorrhage in the stomach from unknown cause. The pia mater of the brain showed some milky thickening in the parietal region. There were no sclerotic or pigmented areas or other focal lesions in the brain. No microscopic examination was made.

Case 3. A man 22 years old developed a tremor of the intention type and a corneal ulcer with hypopyon. As he showed some nystagmoid jerkings and scanning speech a diagnosis of multiple sclerosis was made, but the plantar and abdominal reflexes were normal.

Considerable work was done to determine the nature of the pigment met with in the two necropsy cases. Certain tests suggested that the pigment was silver, but the author himself does not believe so and considers it of metabolic origin. At the end of his article he comments on the similarity of his cases to one reported by

Völsch[9] of a girl 17 years old afflicted with violent tremors, difficulty in speech and swallowing, and contractures of the extremities. There was slight mental impairment and dulness. The plantar and abdominal reflexes were normal. The contractures of the extremities resembled those of hysteria. The patient died of scarlet fever and the necropsy revealed cirrhosis of the liver and chronic enlargement of the spleen, some edema of the meninges, but no gross or microscopic changes in the central nervous system. In this case a diagnosis of pseudo-sclerosis (of Westphal) was made.

[Both the cases of Fleischer and that of Völsch assume considerable interest in view of the just described "Progressive Lenticular Degeneration with Cirrhosis of the Liver," of Wilson. In fact this author in a postscript to his paper calls attention to the case of Völsch which had appeared previously. He considers it possible that lesions of the lenticular nucleus might have been overlooked on account of the way in which the sections were made. The prominence of tremor and mental symptoms in Fleischer's cases and the presence of cirrhosis of the liver make us strongly suspect that his cases were instances of the disease described by Wilson, and the rather meager anatomic description does not convince the critical reader that the lesions of the lenticular bodies may not have been overlooked because they were not looked for.—Ed.]

The Hypophysis.

Cushing's new book, entitled "The Pituitary Body and its Disorders," [1] should be read by anyone who wishes to keep abreast with the rapid strides of the faithful investigators who in recent years have produced such a marvelous array of clinical and experimental data concerning the diseases of this small, hidden and mysterious, yet very essential, organ. The morphology, physiology, chemistry and pathology of the gland are first considered. Among the data adduced are the following:

Normal posterior lobe activity is essential to effective carbohydrate metabolism. An intravenous injection of

---

(9) Deutsche Zeitschr. f. Nervenheilk., Vol. 42, p. 335.

(1) 322 pages, with 319 illustrations. J. B. Lippincott Co., Philadelphia and London, 1912.

posterior lobe extract produces glycogenolysis, and its continued administration in excessive amounts leads to emaciation. A diminution of posterior lobe secretion occurring in certain conditions of hypopituitarism (whether experimentally produced or the result of disease) leads to an acquired high tolerance for sugars, with the resultant accumulation of fat.

An acquired degree of tolerance for sugars is not only of possible value from a diagnostic point of view as a measure of posterior lobe activity, but it also offers a suggestive therapeutic lead for rational opotherapy.

Histologic Studies of the organs in these states of experimental hypopituitarism served to show, what has been found to be true of primary hypophyseal disease in man, that in addition to the fatty changes which are particularly noticeable in the liver, more or less definite alterations occur throughout the whole ductless gland series. After some of his hypophysectomies an hypertrophy of the thyroid was observed by Caselli, and in Cushing's experience the gland in the acute stage of the experimental lesion is often the seat of active hyperplasia, and in the later stages is somewhat enlarged and colloidal. A persistent thymus is often demonstrable. The adrenal medulla is frequently hypertrophic, and the cells of the zona fasciculata often show an extreme fatty degeneration. The genital organs of the female puppies remain infantile and the ovaries show but few forming Graafian vesicles; the testes of the male puppies likewise show the persistence of a pre-adolescent type of tubule and the Leydig cells appear to have undergone a lipoid metamorphosis. Special studies by Goetsch have demonstrated the existence of alterations in the pancreatic islets and in the secretory cells immediately adjoining them.

It appears, therefore, that in consequence of experimentally induced hypophyseal deficiency marked changes occur in the histologic picture of many if not of all of the other ductless glands. It is, of course, well known that alterations occur in the reverse direction; for example, in the hypophysis after castration (Fichera, Tandler, *et al*) or thyroidectomy (Rogowitsch, Herring, *et al*), especially in young animals; and Cushing has observed definite changes in the posterior lobe after pancreatectomy. However, it may be said that the secondary alterations occurring in the ductless gland series seem to be more pronounced when induced by a primary hypophyseal lesion than after corresponding lesions of any other member of the series.

"Our first experiences, therefore, with hypophysectomized adult canines deprived of all but a fragment of the pars anterior disclosed a clinical syndrome with adiposity, increased sugar tolerance, lowered body temperature and reversive sexual changes as its chief features. Later experiences, with the production of corresponding hypophyseal defects in puppies, disclosed a similar syndrome, with a persistence of sexual infantilism and the additional factors of skeletal undergrowth and evident psychic disorders. The latter condition, as corroborated by Aschner and Biedl, a corresponds with the human *typus Froehlich*; the former is the experimental counterpart of what will be recognized in man as an adult form of this syndrome—hypopituitarism originating before adolescence *versus* hypopituitarism originating subsequently.

As to the pathology, the following statement is of importance: "A better knowledge of the alterations which occur under the influence of various physiologic states, such as Erdheim's important investigations have given us for the gland in pregnancy, will doubtless be required before we can attempt to interpret the changes occurring in disease. Aid will come, too, from the study of such alterations in the nature of compensatory hyperplasias as follow partial extirpations. Our studies of the changes which occur in the posterior lobe after the experimental obstruction of the stalk or after inducing an experimental hydrocephalus, lie in this direction and have enabled us to appreciate better than we otherwise could have done the histologic alterations in the pars intermedia and nervosa which occur in many conditions of intracranial disease in man, particularly in those relating to the invasion of the pars nervosa by the investing cells. In every case of increased intracranial tension, from whatever source, there probably occur secondary changes in the hypophysis, often with gross deformations and resultant functional disturbances which frequently elicit recognizable clinical manifestations.

Clinical States and their Classification. Cushing states that at first attempts were made to classify the cases according to whether hyper- or hypo-function affections, but it was soon found that comparatively few could be assumed to exist, analogous to the thyroid cases came strictly under only one of these headings. It is, therefore, best to employ the term "dyspituitarism" for the whole class of functional disturbances of this organ.

Classification of States of Dyspituitarism. "In an analysis of our cases of obvious hypophyseal implication a tentative grouping was first made into (1) those in which the gland was seemingly the primary seat of disease, and (2) those in which it was secondarily involved, usually by the direct compression of an adjoining, or the more remote effects of a distant, cerebral lesion. However, on clinical grounds it often proved impossible to tell, in the first place, whether or not many of the interpeduncular tumors were actually glandular in origin; and, secondly, recognizable hypophyseal symptoms brought about by distant lesion—cerebral tumors, for example—proved to be so uniformly present that this simple subdivision was abandoned in favor of one comprising a larger number of clinical groups—five, as a matter of fact. These groups are as follows:

"Group I. Cases of dyspituitarism in which not only the signs indicating distortion of neighboring structures but also the symptoms betraying the effects of altered glandular activity are outspoken.

"Group II. Cases in which the neighborhood manifestations are pronounced but the glandular symptoms are absent or inconspicuous.

"Group III. Cases in which neighborhood manifestations are absent or inconspicuous though glandular symptoms are pronounced and unmistakable.

"Group IV. Cases in which obvious distant cerebral lesions are accompanied by symptomatic indications of secondary pituitary involvement.

"Group V. Cases with a polyglandular syndrome in which the functional disturbances on the part of the hypophysis are merely one, and not a predominant feature of a general involvement of the ductless glands.

"Under each of the first four groups there will naturally occur three subdivisions; namely: (1) The cases in which the clinical manifestations of past or of existing hyperpituitarism predominate (more particularly overgrowth, resulting in gigantism when the process antedates ossification of the epiphyses—*typus Launois;* resulting in acromegaly when it is of later occurrence—*typus Marie*); (2) those in which the clinical manifestations of hypopituitarism predominate (adiposity, with a persistence of both skeletal and sexual infantilism when the process originates in childhood—*typus Froehlich*); adiposity with sexual infantilism of the reversive form when it originates in the adult—the type we have explained on experimental grounds and of which clinical illustrations are to be given; and (3) the mixed or transition cases exhibiting some features of both states—in other words, with evident dyspituitarism."

The bulk of the work is devoted to a narration of over 50 personal cases, most of which were operated, and many, unfortunately, soon came to necropsy. The case reports are thorough, profusely illustrated, and cover all varieties of primary and secondary affections of the hypophysis. Finally, we have a general treatise on symptomatology and treatment. The consideration of the latter is prefaced by the following general statement:

"From a therapeutic standpoint we are confronted by a variety of problems, some of which call for mere symptomatic medicinal measures, some for operative relief, and some for the administration of glandular extracts to make up for a deficient secretion. It is necessary to determine, therefore, for each case, just what the local conditions may be, and, so far as possible, what is the existent stage of glandular activity. One patient may need merely a sellar decompression to alleviate the headaches of capsular distention, another may require partial extirpation of a struma to relieve chiasmal pressure, another, who has no neighborhood symptoms, may require merely glandular feeding.

"On the other hand, combinations of these measures may be needed. Thus, one and the same individual may urgently require a subtemporal decompression for the relief of general pressure symptoms and subsequently the sellar floor, or even a portion of the large struma, may be removed by a transsphenoidal route to save the chiasm from further damage; at the same time, the *x*-rays may be employed to check further cell division of an active chromophobe struma, the patient meanwhile receiving glandular administration to supply the secretory deficit. Hence, the treatment of these cases is neither strictly surgical nor medical, but various measures must be adopted according to the immediate demands of the individual problem."

The following is a résumé of the surgical treatment: "The operation of choice for the majority of cases, as being less mutilating and yet one which furnishes as wide an avenue of approach as any, is a transsphenoidal operation through a median inferior nasal opening, reached by a sublabial incision and a submucous resection of the vomer, the turbinates being flattened but not removed. The essential precautions are (1) to be correctly oriented in regard to the sphenoidal cells so as to avoid a misdirected approach to the posterior ethmoid region, where a projection of the cribriform plate may suggest to the inexperienced the hypophyseal floor; (2) to be sure of the local conditions by a careful stereoscopic study of *x*-ray negatives and to operate under their guidance; (3) to have perfect anesthesia; (4) to have the courage to withdraw for a second session in case there is any uncertainty as to the character of the tissue exposed after incising the pituitary capsule.

"The operation of second choice—a subtemporal procedure—may be necessary in the case of a superimposed lesion with small sella, or when with an enlarged sella a flattened gland is interposed. As a rule, this means little more than a decomposition, though in favorable cases it may be possible to expose and partially remove a struma or to evacuate a cyst by this lateral method of approach.

"Surgical measures resolve themselves (1) into a sellar decompression (a) for persistent hypophyseal headaches, (b) for the purpose of encouraging the extension of a glandular struma in the direction of the sphenoidal cells rather than into the cranial chamber; (2) into the partial removal of an hyperplastic gland in the active stage of hyperpituitarism; (3) into the partial removal of a tumor or struma for the relief of neighborhood symptoms; (4) into a subtemporal decompression for the palliation of pressure symptoms when an intracranial extension has occurred; (5) into a subtemporal or sellar decompression, or both, to permit of the more favorable and direct application of radiotherapy; (6) into the exposure of the brain or of some other organ in case of marked hypopituitarism, for the purpose of implanting a viable gland.

Infantilism in Pituitary Disease is discussed by A. W. Hewlett,[2] of Ann Arbor, who calls attention to the fact that the symptoms in cases of hypophysis tumor may be roughly divided into two classes: Those due to the mechanical effects of the tumor and those which seem to be due to an altered internal secretion. To the former group belong the headache, vomiting and visual disturbances. The most characteristic of these, primary optic atrophy and unilateral or bilateral temporal hemianopsia, are due to direct pressure on the optic chiasm or nerves. To the latter group belong the sexual disturbances (retarded sexual development, cessation of menses, etc.), the obesity, the stunted growth and possibly the occasional diabetes insipidus. Three cases are reported.

Case 1. *History of brain tumor. Blindness due to primary optic atrophy. Retarded growth. Sexual infantalism. Obesity. Diabetes insipidus.* The patient was a man 27 years old who between the ages of five and nine years had symptoms of brain tumor with severe headache, vomiting and emaciation.. Dimness of vision gradually developed and progressed to complete blindness. He began to suffer from polyuria, and at the age of nine years the headaches and vomiting disappeared and the weight commenced to increase. Since that time he has been extremely obese. He has never showed the slightest indication of sexual development. He had a lumbar lordosis, marked genu valgum and comparatively small hands and feet. The lower jaw was also rather small and the teeth crowded together in both jaws. His height was 4 feet, 9½ inches; his weight, 166 pounds. The large deposits of fat in the mammæ, buttocks, abdomen and above the symphysis pubis, gave the figure a feminine appearance. The genitals were very small. There were only a few coarse hairs in the axillæ and about the pubes. The voice was high-pitched and of a melodious quality. While of good intellect the patient was rather childish in his emotional characteristics. The urine was always increased in amount, sometimes reaching eight or more quarts a day and of a constant specific gravity of 1003 to 1004. Ophthalmoscopic examination showed primary optic atrophy. No good radiograph of the skull was obtained, but the author rightly concludes that there can be no doubt as to the presence of a tumor in or near the hypophysis, producing symptoms which are considered due to deficient function of this organ (hypopituitarism).

---

(2) Archiv. Int. Medicine, January, 1912.

Case 2. *Man 40 years old. Since the age of 13 years periodic headaches with vomiting. Mild sexual infantilism. Adolescent height and figure. Bitemporal hemianopsia with primary optic atrophy. Bony changes in the region of the sella turcica.* From the age of 13 years the patient suffered from headaches, usually associated with vomiting and occurring every month or two. He had had occasional seminal emissions between the ages of 14 and 30 but none since then, and he never had sexual intercourse. The headaches were usually frontal and had increased greatly in severity during the last three or four years. Vision had been failing the last two years. He was a short and slender man, 5 feet, 2 inches tall and weighing 76 pounds. There was almost no axillary and pubic hair and the testicles were small and rather soft while the penis appeared normal. Vision was so reduced that the patient could scarcely count fingers and the fields showed bitemporal hemianopsia.

The blood-pressure was low, ranging from 84 to 87 and only on one occasion was it as high as 110. One to two litres of urine were passed daily with an average specific gravity of 1006 and restriction of liquids for a week did not raise it. *X*-ray examination showed an absence of the shadows normally cast by the bones surrounding the sella turcica. Operation was suggested to the patient but declined on account of the danger and because a restoration of sight could not be promised.

Case 3. *Retarded sexual development. Small frame. Large sella turcica.* The patient was a man 52 years old, 5 feet, 2½ inches tall and weighing 91 lbs. He was of slender build and gave a history of having had an illness at the age of 16 which was diagnosed as spinal meningitis. His first definite sexual feelings and first seminal emissions occurred at about the age of 30, but he never developed normal sexual impulses. A radiograph of the skull showed an abnormally wide sella turcica. The optic nerves were rather pale and there was a slight general limitation of the visual field due to cortical cataract.

THE PINERAL BODY.

Pearce Bailey and Smith Ely Jelliffe[4] report a case of teratoma of the pineal body, describe the symptom-complex of disease of this organ and give an abstract of the entire literature of pineal tumors which now comprises 59 cases. The symptoms may be divided into two classes, neurologic and metabolic.

The neurologic symptoms are, first of all, those of increased intracranial pressure, which are among the first noted. On account of the location of the pineal body pressure on the veins of Galen which drain the choroid plexuses of the ventricles, and on the aqueduct of Sylvius through which the ventricular fluid escapes, is produced early, resulting in internal hydrocephalus to which the increased pressure is due. Headache, choked disc, vomiting, dizziness and drowsiness are early symptoms. Of local pressure symtoms those due to pressure on the corpora guadrigemina and posterior

---

(4) **Archiv. Int. Med., December, 1911.**

longitudinal bundles are frequent, such as ocular palsies and pupillary abnormalities. Paralysis of the upward conjugate movements of the eyeballs is particularly frequent.

The metabolic symptoms to which attention has been particularly drawn by Marburg[5] are (a) adiposis, (b) early sexual maturity, and (c) cachexia. It has not been determined whether pineal tumors alone can produce adiposis but in at least a majority of cases it appears that it is due to secondary disturbance of the function of the posterior lobe of the hypophysis by the hydrops of the third ventricle. It will be recalled that the characteristic symptom-complex of the posterior lobe of the hypophysis consists of adiposis and genital atrophy (Fröhlich's dystrophia adiposo-genitalis). The sexual symptoms usually consist of enlargement of the external genitals, early appearance of the genital hairs, premature sexual desire generally manifested by early masturbation and in some cases early changes in the voice. It is uncertain whether the genital symptoms are due to hyperfunction of the pineal body or to the secondary effect on the hypophysis, although the former appears more plausible because genital precosity is not a common feature of primary hypophysis disease.

The type of tumor may be of some importance as it seems possible that the genital hyperfunction is particularly frequent in cases of adenoma or teratoma, in which a pineal hyperfunction probably exists. Whether the cachexia is a local or general symptom of tumor has not been determined.

The authors' patient was a boy 12 years old who entered the hospital on account of failing memory and difficulty in walking, the clinical picture at first suggesting juvenile paretic dementia. He was fat and large for his age. He staggered in walking, complained of heaviness of the head rather than severe headache, was drowsy and had difficulty in concentrating his attention. Choked disc with impaired vision, contracted fields and interlacing of the color fields were present. The upward eye movements were completely lost and the outward mobility was moderately impaired in both

---

(5) Practical Medicine Series, 1910, Vol. X, p. 95.

eyes. The pupils reacted sluggishly to light and in accommodation and occasionally nystagmus was present. The reflexes in the legs were increased with ankle-clonus and Babinski's sign. There was ataxia of both arms and legs. A decompression operation was performed over the left posteriot fossa without relief and the patient died two weeks later. Necropsy revealed a large rounded encapsulated tumor one and a half inches in diameter replacing the pineal body (Plate XI). The tumor was soft and consisted of sebaceous matter, hair and pieces of cartilage; hence it was a teratoma. The corpora quadrigemina were flattened and the aqueduct of Sylvius was almost obliterated. The ventricles were greatly distended. It is interesting that a shadow in this region was noted in the *x*-ray picture which is explained by the cartilage in the tumor.

DISEASES OF THE SPINAL CORD

ACUTE ANTERIOR POLIOMYELITIS.

Mode of Infection. Simon Flexner[1] calls attention to the fact that at the present we possess only one means of detecting the virus and that consists in its transmission to monkeys in which it produces characteristic paralysis and anatomic changes. By this method the virus has been found to exist constantly only in the brain, spinal cord and mesenteric lymph-nodes. It has also been detected in the mucous membrane of the nose and throat, in the secretions of this membrane and in the mucous secretions of the stomach, small and large intestine. The virus has not been detected in such important organs as the spleen, kidneys, liver or bone-marrow. It is not unlikely that on account of the complicated method necessary for its detection small quantities of virus may escape discovery.

It is noteworthy that the distribution of the virus is identical in the spontaneous disease of man and the experimental disease of animals. Nor does it matter in what manner the experimental inoculation is accomp-

---

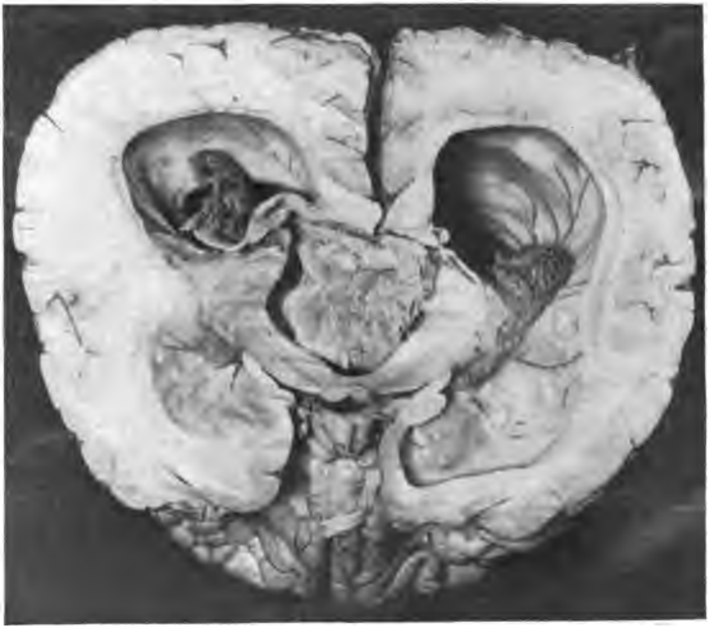
(1) Jour. Amer. Med. Assoc., Oct. 12, 1912.

lished or whether the virus is introduced by injection into the brain, the large nerves, or the subcutaneous tissue, or the peritoneum, or whether it is merely applied to the nasal mucous membrane. We know that the virus escapes from the body with the secretions of the nose and throat and the discharges from the intestine. Flexner believes that after the virus has found its way to the meninges, it does not long remain in the cerebrospinal fluid. Part of it escapes into the blood in which it does not appear to undergo any further increase in amount and indeed seems even incapable of surviving long.

Another part escapes regularly by way of the lymphatic channels surrounding the short olfactory nerves and reaches the mucous membrane of the nose whence it may escape into the secretions, pass into the mouth and be swallowed, or it may become established in the substance of the mucous membrane where it undergoes an increase. The swallowed virus seems regularly in part to find its way to the mesenteric lymph-nodes. Flexner considers it settled that in man the infection occurs by way of the nose. It is known that in monkeys the virus is incapable of passing the barrier of the unbroken or slightly abraded skin, or of being taken up from the stomach or intestine unless the functions of these organs are previously disturbed and arrested by opium, and it is further known that it traverses with difficulty, or even not at all, the substance of the lungs. On the other hand it is established that the virus passes with readiness and constancy from the intact or practically intact mucous membrane of the nose to the central nervous system, and that this membrane is the best place to inoculate next to the brain itself. It therefore is seen that the nasal mucous membrane is the site both of ingress and egress of poliomyelitis in man.

Flexner also points out that the clinical evidence is strong that human carriers exist. (See the report of Swedish Investigators on a later page.) As to the possibility of insect-carriers it has been shown that house-flies can act as contaminators since the virus survives on the body and within the gullet of these insects. Howard and Clark attempted unsuccessfully to infect

PLATE XI.



Teratoma of the pineal body.—Bailey and Jelliffe. (Page 109.)

the common varieties of mosquitoes and the body- and head-louse, but succeeded in producing infection in bedbugs which were made to feed upon the blood of inoculated monkeys. The virus remained alive within these insects for a period of many days. The inoculation of monkeys with a filtrate prepared from them gave rise to characteristic paralysis and anatomic lesions. This result is significant since it shows that insects are capable of taking up the virus from the blood in which it exists in minimal quantities and in harboring it for a considerable period in an active state; but it does not show that multiplication occurs within them or that in nature they act as the agents of inoculation.

Dust as a Carrier of the Infection. M. Neustaedter,[2] of New York, long ago became convinced of the rôle of dust in the spread of poliomyelitis. He says: "For a period of eleven years I have had the opportunity to examine school children of ages from 6 to 14 years. I have noticed that between the ages of 5 and 8 years there are present hypertrophied tonsils in 85 per cent. of the children examined, but as we go up in the age-scale, say at 14, we find only about 15 per cent. of the children with hypertrophied tonsils. The question naturally arises, what has become of these tonsils? My explanation is that these children of from 5 to 8 years of age are forced to breathe the air nearest to the ground, which is more full of solid particles than the upper strata of atmosphere, and that these solid particles are constantly irritating the nasopharynx and producing the condition described. As the child grows taller and is inhaling air of a lesser density so far as dust particles are concerned, the irritation subsides and the tonsils atrophy."

He collected the sweepings of rooms occupied by poliomyelitis patients and macerated them in sterile water and then filtered them through porcelain. The filtrates were inoculated into monkeys, with positive results, typical poliomyelitis being produced. The author believes that the virus finds its way into the dust by way of the nasopharyngeal discharges and once in the atmosphere, the virus may be disseminated in the fol-

---

(2) Jour. Amer. Med. Assoc., Sept. 7, 1912.

lowing manner: (1) by direct contact; (2) by healthy carriers, as human beings, on clothing or by domestic animals; (3) by winds scattering the dust; (4) by bathing in stagnant water in an infected neighborhood.

He concludes by recommending the following measures, some of which, however, as was pointed out by Dana in the discussion, are difficult of execution in many communities.

1. The streets of a neighborhood where cases occur ought to be washed thoroughly and oiled daily.
2. The floors and trimmings of an apartment where a case occurs must be scrubbed daily with an antiseptic solution.
3. Everyone occupying this apartment should have his nasopharynx sprayed with hydrogen peroxid, to be followed by a spray of salt solution. This procedure applies equally to the patient.
4. A strict quarantine should be maintained for at least two months.
5. Bathing in stagnant water in a neighborhood where a case occurred should be prohibited, as well as playing around sand heaps.
6. Domestic animals should be thoroughly disinfected, thoroughly bathed and scrubbed with an antiseptic solution, and removed from premises.

The Rôle of Flies in the Transmission of Poliomyelitis was discussed by M. J. Rosenau,[3] of Boston, before the *International Congress of Hygiene.* When he began the study of the transmission of the disease he favored the idea of direct contagion but his faith was shaken after 18 negative results in attempting to prove the presence of the virus in the secretions from the nose and throat. Other observers, with the exception of those in Sweden (see subsequent pages) have had similar negative results. Another striking fact was the rapid spread in thinly populated rural districts while few cases occurred among children crowded in city schools, institutions and tenements. The resemblance of the lesions of this disease to rabies suggested the possibility of some kind of wound infection. Rosenau decided to concentrate his attention on the stable-fly,

---

(3) Jour. Amer. Med. Assoc., Oct. 5, 1912.

Stomaxys calcitrans, which is a vicious and voracious insect, present everywhere but much more common in the country than in cities. A number of these flies were allowed to bite infected monkeys in all stages of the disease. Then these flies were allowed to bite 12 healthy monkeys, 6 of which developed the typical disease, and 3 others apparently a mild infection. Three of the affected monkeys had diarrhea, and on the whole the disease produced in this manner bore greater resemblance to the type seen in children than to that produced by cerebral inoculation.

The author merely records his experiments and then says: "I have purposely refrained from drawing any conclusions. It is clear we have had infection which has been transmitted through the agency of these flies from monkey to monkey. It requires several weeks before the fly is able to infect every monkey so far as we are able to tell at the present time. What reference this has to the human cases I am not able to say. If I were a health officer of a community and poliomyelitis prevailed, I should institute a fly campaign."

The work of Rosenau has been confirmed by the results of similar experiments by Anderson and Frost[4] prepared under conditions as nearly similar as possible. On October 3d a monkey was inoculated intracerebrally with known poliomyelitis virus. Two hours afterwards the infected monkey was exposed to the bites of about 300 stable-flies (*Stomaxys calcitrans*), and the same flies were allowed to bite it daily until its death on October 8th. It developed paralysis on October 7th. They were also allowed to bite another monkey inoculated on October 5th which developed paralysis on October 9th and died the same day. Beginning October 4th two healthy monkeys were exposed daily for about two hours to the bites of these same flies, and October 5th a third fresh monkey was added. All three developed the disease, the first two on October 12th, the third one on October 13th—eight days after the first exposure to the flies. So far one of the cords has been examined and showed the typical lesions of poliomyelitis. Material from the cord of this monkey

---

(4) Public Health Reports, Vol. 27, Oct. 25, 1912.

was inoculated into the brain of a fresh monkey on October 14th. This animal became paralyzed on October 17th, and died the next day, showing minute hemorrhages and evidence of pathologic changes in the gray matter of the cord.

The Visceral Lesions of Human Cases were studied in four necropsy cases in children by Flexner, Peabody and Draper.[5] "All the fatal cases examined by us showed some, and usually a high degree of hypertrophy of the lymphoid tissues. The affection of these tissues was widespread, if not universal, and included the tonsils, small intestine, thymus and the superficial and deep lymphatic glands. The glands about the trachea and bronchi and those of the abdominal cavity were usually much enlarged and definitely softened in consistence. The mucosa of the large intestine escaped affection, although the mesocolic glands did not. While the cervical, axillary and inguinal nodes do not become prominent, they all showed definite enlargement to the naked eye. The spleen was somewhat enlarged and the appearance was universally altered. The Malpighian bodies were prominent, and the pulp was increased and of dark or grayish hue. Defects in the intestinal mucosa were never observed. The necropsies were performed a few hours after death, so that the organs were secured in a state free from post-mortem changes."

The authors are led to place the disease among the affections in which the viscera in general are affected. The main injury appears to be inflicted on the central nervous system, next on the lymphatic organs, and lastly on the parenchymatous organs. Of the latter the most pronounced changes were found in the liver, consisting of focal necrosis of the liver cells. The number of areas of necrosis is very large and their extent varies from a few cells to an eighth of a lobule.

The Histologic Changes are thoroughly discussed and profusely illustrated by Richard Walter[6] on the basis of four cases which were subjected to a very detailed examination. His remarks on the much-discussed question whether the inflammation in the pia is primary or sec-

---

(5) Jour. Amer. Med. Assoc., Jan. 13, 1912.
(6) Deutsche Zeitschr. f. Nervenheilk, Vol. 45, p. 79.

ondary, is of interest, especially to those who have followed the controversy between Wickman of Sweden and Harbitz and Scheel of Norway on this point. Walter, in his cases, found conditions like those described by Wickman. The infiltration was most marked in the anterior part of the pia. He never saw an extension of the meningeal inflammation to the nervous tissues. He agrees with Wickman that the myelitic changes are primary because he often encountered sections of the cord in which there was marked inflammation in the gray matter, while the pia was not involved. He rejects the hypothesis advanced by Harbitz and Scheel that the inflammation in the meninges is primary, and that the cord is infected from the meninges along the vessels.

Walter describes minutely the various types of inflammatory cells met with and discusses their significance. His findings in the various internal organs were similar to those described by the Swedish authors quoted elsewhere in this volume. He found enlargement of the spleen, the pulp of which was soft and hyperemic; marked swelling of the mesenteric glands and the intestinal follicles; redness and swelling of the mucosa of the intestine. In one case he found small inflammatory foci around a bile-duct within the liver, and in another case marked fatty degeneration of the liver. In one case inflammatory foci were found in the heart muscles and in the epicardium. The kidneys in one case showed slight glomerulitis.

Sensory Disturbances. It was formerly thought that acute poliomyelitis affected only the anterior horns, and that sensory disturbances never occurred. From the recent epidemics we have learned that the inflammation also attacks the meninges and the white matter, and cases with the pictures of transverse myelitis with loss of sensation have been known to occur. Strümpell[7] reports a peculiar case in which he assumes an involvement of the posterior gray horns of the cord and designates the disease type as "acute posterior poliomyelitis."

[This term is perfectly proper and logically should be applied to inflammation of the posterior horns in contradistinction to the ordinary form affecting the anterior

---

(7) Deutsche Zeitschr. f. Nervenheilk, Vol. 45, p. 124.

horns, but unfortunately, for the lack of a name for inflammation of the spinal ganglia and the homologous ganglia on the cranial nerves, J. Ramsey Hunt has already appropriated the term "acute posterior poliomyelitis" for the herpetic inflammation of these ganglia. The subject was discussed in the *Practical Medicine Series,* 1910, Vol. 10, pp. 156 and 158. It would be well if this confusion in nomeclature could be straightened out before it becomes worse confounded. To call inflammation of the geniculate ganglion on the facial nerve poliomyelitis certainly appears unreasonable.—Ed.]

The patient was a boy 19 years old who was taken ill with general malaise and disagreeable sensations in the right side of the chest. Within a few days both hands and both legs became so weak that he could not stand or walk. There was neither pain nor sphincteric disturbance. After four weeks the paralysis in the legs disappeared and the left hand became strong again in six weeks, but the right hand remained partly paralyzed. He was admitted to the clinic six weeks after the acute attack and then presented marked atrophy and paralysis in the right hand and forearm. The tendon reflexes of both arms were not obtained while those in the legs were present. In short the muscular condition was typical of an old poliomyelitis.

In addition, however, the patient presented a striking sensory disturbance, namely, on the right side a zone of loss of pain and temperature sense which extended anteriorly from the third rib to the ensiform cartilage and passed slightly upwards posteriorly so as to reach the spinal column two inches higher than in front. On the rest of the right side of the body sensation was normal while on the left side the same type of sensory disturbance extended downward from the level mentioned, involving the entire body half below this level.

Tactile sensation was not affected. The patient remained in the hospital for two months during which time the condition was entirely stationary. Strümpel believes that the case was undoubtedly one of typical acute poliomyelitis with the greatest degree of inflammation in the anterior horns of the eighth cervical and first dorsal segments. The distinct sensory dissociation present indicates that the cause of the sensory disturbance was extension of inflammation to the posterior horns at a somewhat lower level than the maximum involvement of the anterior horns. In connection with this case Strümpel discusses at considerable length the present state of our knowledge of the conduction of sensation.

As to observation of sensory involvement in acute poliomyelitis, E. Müller in his monograph, published in 1910, stated that he has frequently found hypesthesias in the early stages and in one case the pain and temperature sense were lost in the paralyzed arm. Wickman observed a patient who still showed distinct loss of pain and temperature sense in the legs one year after the onset of paralysis, and P. Krause[8] observed an almost exactly similar case.

A Clinical Study of Poliomyelitis, is the title of a monograph by Peabody, Draper and Dochez issued by the *Rockefeller Institute* (dated June 24, 1912). It is based on 161 cases observed during 1911 and 22 cases from previous years. The whole subject is treated in a thorough manner. Of particular interest are the results of the study of the blood and of the spinal fluid. These investigators found a constant and marked leukocytosis, the count being as high as 30,000. Leukopenia was present in one case only. Besides the increase in the total number of cells there were found an equally constant increase of polymorphonuclear cells of 10 to 15 per cent. and diminution of lymphocytes of 15 to 20 per cent.

The spinal fluid was examined in 69 cases, 233 samples being obtained. In 43 cases the fluid was obtained in the first week of the disease; in 4 cases in the preparalytic stage, and in 2 abortive cases. The methods used for the examination of the fluid included estimation of the pressure, determination of the number of cells present, estimation of the chlorids, observations of the power of reducing Fehling's solution and estimation of the globulin-content. Globulin was tested by the butyric acid method of Noguchi. In nearly every instance the fluid was clear and colorless, only a few showing a very slight opalescence. In a few cases,

---

(8) Deutsche med. Wochenschr., 1909, p. 1823.

chiefly early ones, a delicate web-like clot formed, but it bore no apparent relation to the globulin-content of the fluid. The pressure determinations were unsatisfactory as most of the patients were crying children. The chlorid determination showed no marked deviation from the normal. Every fluid examined possessed the power of reducing Fehling's solution which is a normal condition. According to these authors the reducing substance may be absent in meningitis and this fact may be of diagnostic value.

Nearly all of the cells encountered were mononuclear. In a few very early cases, including those in the preparalytic period, polymorphonuclear cells predominated. Of the 43 cases examined in the first week 38 showed cell-counts above normal. (The authors regard as many as 10 or 12 cells as normal.) In 23 of these cases there were over 50 cells per c. mm. Two cases examined in the prodromal period showed 990 and 650 cells respectively. Of 45 cases in the second week, 8 cases gave a count of over 50 cells and in 23 cases the count was above normal. Of 40 cases in the third week only one gave a count over 50, and only 8 above normal. Six cases in the first week, 22 cases in the second week, and 32 of the 40 cases in the third week showed normal counts.

The globulin-content is usually low in the first part of the acute stage, especially as compared with the cell-count. It rises during the second and third weeks and then gradually falls, though frequently globulin is present long after all acute symptoms have passed. A strong globulin test was obtained on the forty-sixth day of the disease but only 6 of 43 cases showed a distinct reaction during the first week. During the second week, 20 out of 45 cases showed a positive reaction, in the third week 16 out of 40, and in the fourth week 9 out of 30. In 11 cases the fluids were followed until both cell-count and globulin reaction became normal. The return to normal occurred in the third week in two cases, in the fourth week in two cases, in the fifth week in one case, in the sixth in two cases, in the seventh in three cases, and in the tenth week in one case. The commonest type of fluid seen in the whole series was one with a normal or slightly increased cell-count and a well marked globulin reaction.

"As regards early diagnosis, the cases examined before the onset of paralysis are of the greatest interest. Six cases were seen in what may be termed the prodromal period. Four of these developed paralysis later, and two proved to be of the abortive type. In one case a single lumbar puncture was made on the fourth day after the onset. The fluid gave a cell-count of 650 per c. mm., 90 per cent. being polynuclears, and a positive globulin reaction. Three days later the child suddenly developed paralysis, and in five hours died of respiratory failure. A second case, the sister of a child already in the hospital, was seen on the day after the onset of symptoms. The spinal fluid contained 990 cells per c. mm., 90 per cent. of which were polymorphonuclears, and the globulin reaction was slightly positive. On the following day one leg was paralyzed and the cell-count of the spinal fluid was 627 per c. mm., with 80 per cent. mononuclears. On the next day both legs were paralyzed and the cell-count was 1,221 per c. mm., with 92 per cent. mononuclears, including many large phagocytic cells. The globulin reaction was positive. Thus two cases seen very early in the course of the disease showed fluids with unusually high cell-counts, and with a marked predominance of polymorphonuclear cells. In one of them the change of the cell-picture from the polynuclear type to the mononuclear type could be followed coincidently with the onset of paralysis. Three other cases in the prodromal period, one an abortive case, and two becoming paralyzed on the day of the first lumbar puncture, gave cell-counts of 37, 94 and 113 per c. mm., with 100 per cent., 93 per cent. and 83 per cent. of mononuclear cells respectively, and globulin reactions which fell within normal limits. Finally in one abortive case lumbar puncture on the second day after the onset of symptoms gave a wholly normal fluid. On the third day the count was 62 per c. mm., all being mononuclears, and a slightly positive globulin reaction. On the eleventh day the spinal fluid was again normal. That this was, indeed, an abortive case of poliomyelitis was later rendered most probable by a test showing that the patient's blood-serum was capable of neutralizing active virus when mixed with it and injected intracranially into a monkey.''

The following are the conclusions reached:

''The spinal fluid from cases of acute poliomyelitis during the first few weeks after the onset of symptoms shows, in the great majority of instances, deviations from the normal.

''Fluids taken during the early days of the disease and especially before the onset of the paralysis tend to show an increased cell-count with a low or normal globulin content. At this early stage the polymorphonuclear cells may amount to 90 per cent. of the total. Most fluids, however, show almost exclusively lymphocytes and large mononuclear cells.

''After the first two weeks the cell-count usually drops to normal, or nearly normal, and there is frequently an increase in the globulin content. A slight increase in globulin may persist for seven weeks or longer.

''Analogous changes may be found in the spinal fluid of abortive cases. All fluids examined reduce Fehling's solution.

''The examination of the cerebrospinal fluid in acute poliomyelitis, while giving, as far as is yet known, no specific diagnostic criteria, is of the utmost value as an aid to diagnosis both in preparalytic and in abortive cases.''

As to prophylaxis the following strong plea is made for the institution of strict quarantine: ''Recent investigations which show that in some epidemics the number of families or houses with more than one case may reach 40 per cent.; that persons have been attacked by poliomyelitis several days after a short and single contact with a patient; that markets, fairs and public gatherings may be the means of spreading the disease; that transmission by fomites probably occurs; that the virus may exist in the saliva and nasal mucous membrane in monkeys and in the tonsils in man; and that it may also be found in the dust from the rooms of patients have convinced public health authorities that poliomyelitis should be treated like any other definitely contagious disease.''

It is as yet impossible to determine how long quarantine should be maintained. The Nebraska epidemic in 1909 was apparently checked by imposing an absolute quarantine for three months on all members of a patient's family, with the exception of the breadwinner; and the *Paris Academy of Medicine* recommended to keep the affected child away from school for three months. The authors believe that practically it suffices to isolate the children for four weeks and then when possible to keep them apart from other children for several weeks more.

Treatment. On account of the marked hyperesthesia and pain on moving the limbs the patient should be handled as little and as gently as possible, and the bedclothes should be raised so as not to cause pressure. A light, well-padded splint often helps to steady the limbs and one of these patients used to beg to have the splint replaced when it was taken off. Applications of heat are very useful for overcoming pain. The authors have not become convinced that lumbar puncture is of any therapeutic value. Artificial respiration may possibly sometimes tide the patient over the dangerous period during a temporary affection of the cells controlling the respiratory muscles. Splints, sand-bags, and other apparatus are useful in preventing deformities and usually need be applied only at night so as to leave the limbs free to move during the day.

It is recommended to begin with massage as soon as it is tolerated. But most important of all is active movement or muscle training. With little children it is often easier to get them to use their limbs while they are in a warm bath.

The Swedish Epidemic of 1911 is one of the largest on record, as about 3,800 cases were observed. An exhaustive study of the epidemic was made by the *Bacteriologic Department of the State Medical Institute of Sweden,* and the results are related in a report of 270 pages, in English, presented to the *15th International Congress of Hygiene and Demography* which met in Washington in September, 1912.(9) The report is divided into a number of papers.

---

(9) Investigations on Epidemic Infantile Paralysis, Published by the State Medical Institute. To be obtained through Nordiska Bokhandeln, Stockholm.

I. *The Presence of the Microbe of Infantile Paralysis in Human Beings,* by Carl Kling, Alfred Pettersson, and Wilhelm Wernstedt. In 14 fatal cases material from (a) the mouth and nose, (b) trachea, (c) intestine, and (d) spinal cord was passed through Heim and Berkefeld filters and inoculated into monkeys. The mouth and nose contained active virus in 7 of 11 cases tested, the trachea in 8 of 10 tested, while 2 were uncertain and 1 negative. The cord gave positive results in all of the 9 cases tested.

From 12 living patients material was secured from the mouth, nose and pharynx, and from the large intestine, filtered through a Heim filter and injected into the peritoneal cavity and the sciatic nerve of monkeys. From the mouth and nose positive results were obtained in 7 of 12 cases and from the intestine in 9 out of 10 cases. This very high percentage of successful inoculations, considering the difficulties in infecting the monkeys, leads the authors to conclude *that the virus in all probability is always present on the mucous membranes of the nose, mouth, pharynx and intestine of persons affected by infantile paralysis during the acute stage of the disease. It is obvious that the knowledge of this fact is of the greatest importance in formulating our views regarding the mode of transmission of the disease.*

Extensive inoculation experiments were also made with the secretions from the mucous membranes of so-called abortive or light cases in patients who had been known to have been in contact with cases of poliomyelitis and from patients without any history of such contact. Furthermore similar tests were made with a number of healthy persons living in infected communities. In one family one boy of three years had typical poliomyelitis, and a girl of eight years and a boy of five years had a few days of fever and vomiting with complete recovery, while the father and mother were not infected. Material from the mouths of the parents, the boy of five, and the girl was injected into monkeys with positive results, both in the case of the healthy father and of the girl with the transient indisposition. In the case of the boy the result was doubtful as the monkey used had advanced tuberculosis. The interesting point is that of these three individuals with active virus in their mucous membranes, only one had distinct paresis, while one was an abortive case and one a healthy carrier.

Another family, consisting of father, mother and six children, had been in frequent contact with the other family, and the parents and two children were taken ill with brief fever but only one child developed paralysis.

In a household consisting of father, mother, two children and a domestic, the two children were taken ill with fever three days apart, the first one developing paralysis of one arm, while the other had no paralysis. From the second child material from the mouth and from the intestine was obtained on the fourth day and inoculated into monkeys with the result that one of the two monkeys inoculated with intestinal material had a transient weakness of the arms, while the other developed no symptoms. The monkey inoculated with mouth material also remained well. Three and a half months later two monkeys were again inoculated, one with material from the mouth and the other with material from the intestine of the same child, and both monkeys died and presented evidence of poliomyelitis. In another family one child died of poliomyelitis, and two other children and the father had transient fever without paralysis. Material from the mouth from one of the children with the abortive disease was inoculated into the peritoneum and one sciatic nerve of a monkey which died in six days of poliomyelitis.

Positive results were also obtained with the mouth secretion of three persons with transient symptoms who gave no history of contact with known cases. Most significant of all is the result of experiments made with healthy persons belonging to six families, in each of which only one case of typical disease had occurred. In each family virus-carriers were found. In one household consisting of father, mother, one child and two servants the child developed a mild poliomyelitis while the others remained well. Material from the father and the two servants produced the disease in monkeys, and in the mother's case the test failed as the monkey used died of bacterial peritonitis. In another family 3 carriers were also found, in one 2 carriers and one each in the other three families.

Investigations with a view of ascertaining how long the microbe of infantile paralysis remains on the mucous membranes after recovery were made on a large scale and led to the conclusion that the secretions from the mucous membranes of the mouth and intestine of persons who have recovered from poliomyelitis may give rise to fatal poliomyelitis in monkeys for several months, in one case 204 days, after the onset of the disease.

II. *Experimental Investigations with the Object of Determining the Possibility of Transmission of Infantile Paralysis by means of Dead Objects and by Flies,* by Arnold Josefson. He begins by pointing out that while much speaks in favor of direct infection, the objection has been raised that there has been no report of the disease spreading to other patients or to nurses or to attendants in hospitals. However, the author is now able to report the case of a nurse who died of typical poliomyelitis after she had taken care of a case. In Christiania several nurses were attacked and two died. [These cases were reported by Professor Axel Holst in the Symposium on Poliomyelitis at the International Congress in Washington. He stated that 1,250 cases had occurred in Norway in 1911 with a mortality of 8 to 10 per cent., and the period of incubation was usually 1 to 3 days, but sometimes longer, not infrequently 8 to 10 days and in one case, a prisoner in solitary confinement, the incubation must have been several weeks, possibly 3 months. Here also healthy carriers were demonstrated.—ED.]

In order to determine whether dead objects play a rôle in transmission several experiments were made. Handkerchiefs, picture-books and pieces of fancy work used by patients were allowed to dry in empty sterile vessels for some days and then placed in physiologic salt solution which was passed through a Heim filter and then inoculated into monkeys, and positive results were obtained. Similar experiments were made with flies caught by the patients in the wards, but the results were negative. The author refers to the observation by Landsteiner, Levaditi and Pastia[1] that the virus remains active in sterile milk or sterile water at ordinary room-temperature and light for at least 31 days.

III. *Conclusions with Regard to the Manner of Dissemination of the Infection, and the Possibility of Transmission of the Infection by means of Blood-Sucking Animals,* by Carl Kling, Alfred Pettersson and Wilhelm Wernstedt. These authors do not believe that transmission by insects plays any important rôle though they admit that it may play a part in the origin of house epidemics. They collected fleas in houses in which there were cases of poliomyelitis and ground the fleas in a mortar with salt solution and injected monkeys, with negative results. As to the theory that flies may carry the disease, they point out that in Sweden numerous cases have occurred in winter when no flies had been around for months. Thus, shortly after New Year, 1912, a large number of cases occurred in the northern part of Sweden, on the 66th degree of latitude, that is, practically at the Arctic circle. They agree with Wickman that the disease spreads from one person to another.

Great stress is laid on the part played by abortive cases and healthy carriers. According to the experience of these authors, the virus-carriers and the abortive cases in a family may be four or five times as numerous as the cases showing typical symptoms. "The abortive cases are naturally, in their turn, surrounded by other abortive cases and virus-carriers. Under such conditions it is not surprising that we have up to the present time not succeeded in finding any connection between the different cases. In order to be able to follow the path of the infection, some traces of it must first be found, and it is just this that has hitherto proved impossible. When we discover methods that admit of an etiologic diagnosis instead of the symptomatologic the fog now covering the unknown in infantile paralysis will disappear and allow us to see clearly the mode of infection. *Then we shall probably find that infantile paralysis is a common disease of childhood, generally*

---

(1) Annal. de l'Institut Pasteur, 1911, p. 805.

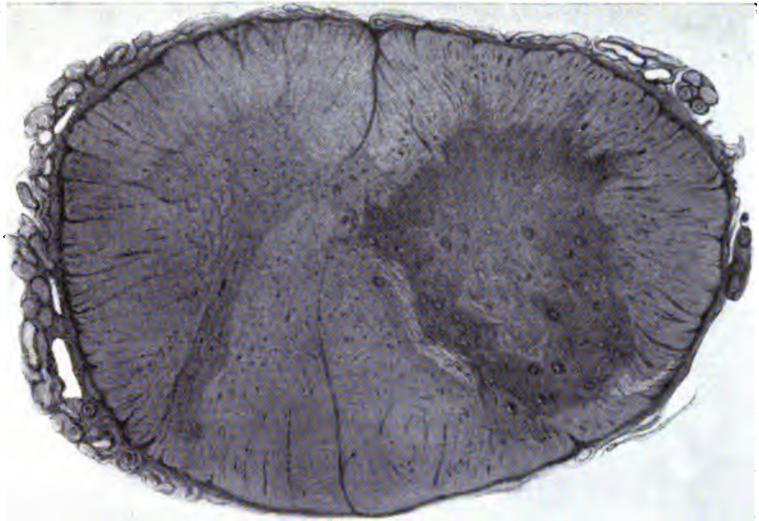
displaying only very slight symptoms, but sometimes acquiring a more virulent character giving rise to disturbances in the nervous system. The same applies also to the other infectious diseases. Scarlet fever, for instance, in certain epidemics, shows an inclination to cause changes in the kidneys or other organs. We shall then, perhaps, find the circumstance that grown-up persons only very seldom are attacked by infantile paralysis explained (as it is in scarlet fever) by the fact that adults have already had the disease in their childhood. Finally, we may discover that the reason why greater towns seem to have been relatively spared the disease lies in the fact that the non-malignant form, which we have hitherto never taken into consideration, is, owing to more lively communication, much more common in the towns than in the country, in this respect resembling a number of other diseases, *e. g.*, measles."

It has been found that animals recovered from the disease have acquired an immunity, and experiences from epidemics in man substantiate this. The disease evades places which have recently been infected. Thus the localities most seriously affected in the Swedish epidemic of 1905 escaped the disease in 1911.

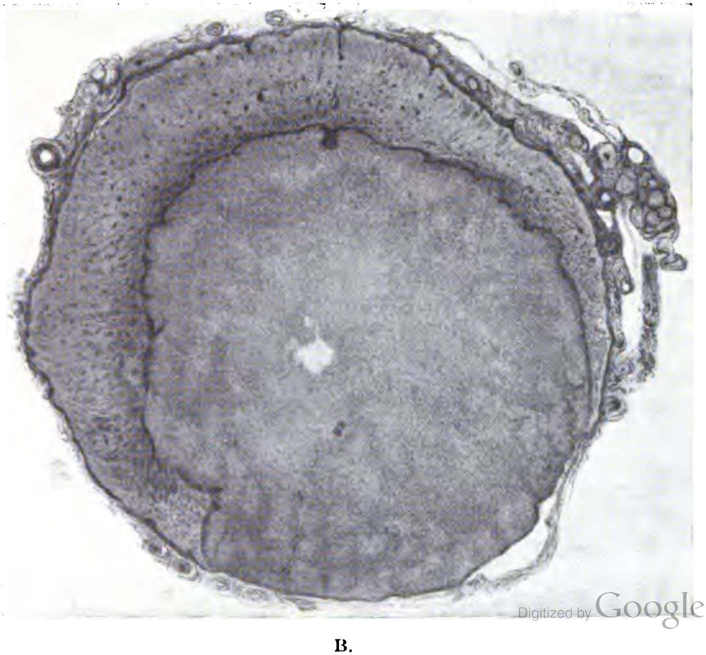
IV. *The Clinical Picture of Experimental Poliomyelitis in Monkeys*, by Carl Kling, Alfred Pettersson and Wilhelm Wernstedt. The material comprised 120 monkeys: The period of incubation averaged 9.4 days. In 3 cases it was as short as two days, and in 23 cases between two and five days. This result coincides very exactly with that of Römer who arrived at the figure of 9.5 days as the average. The symptoms were those usually observed. The mortality was extremely high, being 96.6 per cent., while Flexner and Lewis had a mortality of 75 per cent. and Römer 76 per cent.

V. *The Histologic Changes in the Spinal Cord of Monkeys Affected with Poliomyelitis*, by Carl Kling, Alfred Pettersson and Wilhelm Wernstedt. While their findings in general agreed with those of previous observers they lay greater stress on the degeneration of the ganglion cells themselves, which was occasionally present where the inflammatory cell infiltration was minimal. The glia cells also were found to show distinct changes. It is interesting that the cell infiltrations

PLATE XII.



A.



B.

Tuberculosis of the spinal cord A Case 1 B Case 2—

were relatively less marked in cases infected with fluid from the mucous membranes, which fact is interpreted as evidence of lessened virulence on the part of the virus.

VI. *Histologic Examination of those Mucous Membranes which, on account of the Presence of the Micro-Organism of Infantile Paralysis, may be Suspected to be the Point of Entrance of the Virus,* by Carl Kling and Alfred Pettersson. The changes in the organs in question were not marked but nevertheless were of interest. Thus the tonsils in places showed degeneration of epithelium and round-cell infiltration. In one case in which the disease commenced with a sore-throat the exudation was greater and there were many polymorphonuclear cells. In 4 out of 75 cases examined the salivary glands showed some lymphocytic infiltration and there was catarrhal inflammation of the trachea and bronchi. In 4 cases the solitary follicles of the intestine as well as the mesenteric glands were swollen.

On account of the peculiar character of the catarrhal inflammation of the trachea and bronchi the authors believe that this is the expression of some irritation due to the specific virus, but this does not justify any conclusion as to the method of invasion as the virus is known to exist in the nose, mouth and pharynx, and consequently greater or smaller quantities of it are constantly being swallowed. Through the investigations of Flexner, Clark, and Dochez it is known that the virus can pass through the stomach in a more or less undamaged condition. The changes in the intestine may very well be secondary, and be due only to a constant irritation of the intestine caused by the swallowed virus.

VII. *Some Epidemiologic Experiences from the Great Epidemic of Infantile Paralysis which Occurred in Sweden in 1911,* by Wilhelm Wernstedt. The first cases were observed in March, and in the beginning of April an epidemic of 40 cases occurred in the northern part of the country. The number of cases in the various months is of great interest in view of the present discussion of the possible rôle played by insects. The 3,840 cases which were reported and controlled during the year mentioned were distributed as follows: January, 2; February, 17; March, 12; April, 24; May, 88; June, 83; July, 179; August, 893; September, 1,067; October, 747; November, 483; December, 235. It is thought that the cases did not spread from a single focus to the rest of the country but that the disease originated in several places where sporadic cases occasionally had occurred since the large epidemic of 1905. It is noteworthy that while in 1905 only 7 per cent. of the cases occurred in cities, in 1911, 19.3 per cent. or 741 cases were observed in cities, Stockholm leading with 200 cases, while it had only 10 cases in 1905 when more than 1,000 cases were observed in the country. This phenomenon appears to be only part of the general rule that the epidemic of 1911 spared most of the territory attacked in 1905, as has already been commented on in one of the previous papers. A similar condition has been observed in the epidemics of Norway, Austria and Massachusetts. On the other hand it is noteworthy that nearly all of the new foci were adjacent to territories affected in 1905. Wernstedt also believes in a wholesale immunity on the part of the inhabitants in the places previously attacked.

Poliomyelitis in Norway. Harbitz [2] gives the history of the repeated recent epidemics in Norway. Some interesting observations are related which are strongly in favor of its contagious origin. A physician treated some sporadic cases in his district in the latter part of June and the beginning of July. July 15, he himself became ill with fever, pains in the back, great exhaustion, diarrhea and vomiting. He recovered in three days. July 16, his son, aged 3, was seized with the same symptoms. July 18, his son, aged 5, was stricken in the same manner. He also recovered in three days. July 22, his oldest son was taken with well-defined gastro-intestinal symptoms, stiffness of the neck and paresis of the extremities and face. He recovered in eight days without permanent paralysis. The following days his 1-year-old son, the nursemaid, his wife and her sister were taken sick with acute angina, stiffness of the neck and fever; and finally a neighbor's child, who had been a playmate of the physician's children, was stricken with

---

(2) Jour. Amer. Med. Assoc., Sept. 7, 1912.

symptoms of a genuine poliomyelitis and with subsequent permanent paralysis.

As an argument against the contagiousness of poliomyelitis it has been advanced that nurses who take care of these patients escape the disease. This argument does not hold good; in a separate pavilion of the hospital for epidemic diseases at Christiania were several poliomyelitis patients during July, August and September, 1911. Two of their nurses, aged 28 and 29, became ill in the latter part of August and died with acute poliomyelitis. They had served in this hospital-division for about one month. The virus must, however, have been quite widely disseminated in this hospital, because in August there occurred among the children in the diphtheria pavilion four cases of acute poliomyelitis of which three were in the same ward. In the scarlet-fever pavilion one case of acute poliomyelitis occurred in a child who previously had been taken care of by one of the two nurses who died from poliomyelitis, and finally a nurse who served in one of the medical sections of the same hospital took the disease and died. In this last case it was impossible to trace the source of infection. These cases which occurred almost simultaneously can be explained only by the existence of a direct infection and wide dissemination of the virus among adults and children.

The time of incubation, according to Norwegian experience, has been quite short—two, three or four days, but at times longer. Cases were observed in which a latent condition of the virus must be assumed. An example of this is the case of a young woman who had been in solitary confinement for three months and had contracted acute poliomyelitis. Hers was the only case inside the prison, and it is reasonable to conclude that she came to the institution already infected.

That Norway has taken the lead among all nations in instituting preventive methods is shown by the fact that the following measures were ordered in a circular issued by the National Board of Health in May, 1904:

1. Immediate notification to the board of every case, abortive cases included, with a short history of the disease and its etiology.

2. Isolation of the sick during the acute stage for three weeks.

3. Careful disposition of the patient's excretions and secretions, particularly from the nose and throat. The patient and everybody who comes in contact with him are recommended to use gargles at regular intervals, consisting of solutions of potassium permanganate or hydrogen peroxid. The sick should be furnished with separate dishes, handkerchiefs, pillow-cases, etc.; and anything soiled with secretions or excretions from the patient should immediately be boiled.

4. Disinfection of everything in contact with the patient for a time of three weeks from the beginning of the disease.

5. Particular attention given to the lighter abortive cases and patients with symptoms of angina; also to the possibility of the disease being carried through the intermediary of healthy persons.

6. Attention paid to the possibility of infection through school-children. In much-infected districts it was recommended to close the schools, and healthy as well as sick children from infected homes were prohibited from appearing at school for some time.

These measures received hearty co-operation from the local health authorities and the latter in many instances took additional precautions by prohibiting public meetings.

The Iowa Epidemics of 1910 and 1911 are described by Walter L. Bierring.[3] In the former year there were reported 654 cases with 157 deaths and in 1911, 68 cases with 12 deaths. The final outcome of 343 of the cases in 1910 was ascertained and 131 had made complete recovery without residual paralysis. As to distribution, there was a tendency for the disease to spread along the principal railroad lines traversing the state from east to west, with a predominance for the northern half of the state, which may be explained by the proximity of Minnesota where the disease has prevailed extensively. A very interesting experience was obtained here which is analogous to that noted in Sweden,

---

(3) Interstate Med. Jour., January, 1912.

namely that the localities visited by the disease in 1910 were spared in the following year.

As to modes of transmission the possibility of the disease being spread by means of food and through the medium of dust and insects was carefully investigated, but in no way confirmed. On the other hand, certain facts suggested that domestic animals play a rôle. Thus, a child living 8 miles from the nearest town was taken with the disease but there were no cases in the vicinity and the family had not had any visitors. During the summer many of the chickens on the place had become sick and paralyzed and as soon as this was noticed the farmer would snap off the heads and throw the chickens into the hog-yard. Some time later it was noted that a hog developed paralysis of both hind legs.

In other localities chickens were also observed to have a paralytic disease and some of these animals were submitted to pathologic examination. Thus one chicken was observed in which one wing and both legs were paralyzed and the animal died three days after the onset. In the lower dorsal and upper lumbar regions of the spinal cord a softened area one inch in length was found. Histologic examination revealed cellular infiltrations and hemorrhages like those seen in the human disease. Similar lesions were found in other chickens but efforts to transmit the disease to healthy chickens were not successful.

Treatment. The Value of Hexamethylenamin in early cases constantly receives confirmation. Thus Josefson,[4] of Stockholm, used it in 30 cases, and the 8 in which the treatment was started in the first three days recovered without paralysis. One was treated from the first day, 6 from the second day, and one from the third day. S. J. Crowe[5] enumerates a great many conditions of infection of various parts of the body in which this drug has been effective, among them being common "colds," meningitis, and infection of the ears, genito-urinary and biliary tracts. It has been found the concentration of the drug reaches its height in the cerebrospinal fluid from one-half to one hour after its administration.

---

(4) Hygiea, 1911.
(5) Bull. Johns Hopkins Hosp., September, 1912.

Crowe has seen very few instances of toxic symptoms, although 200 to 700 grains daily for four or five days have frequently been given. The drug should be well diluted, even 10-gr. or 15-gr. doses should be taken in at least eight ounces of water. As it is especially difficult to get sick children to take so much water, one may dissolve 50 to 100 gr. in a quart of salt solution and let it flow slowly into the rectum. Or some of the drug may be added to all of the liquid nourishment of the patient. In the last-named manner 60 to 100 gr. may be given in a day even without the patient's knowledge, because the drug is practically tasteless.

TUBERCULOSIS OF THE SPINAL CORD.

This is a rare affection (tuberculous meningitis is not included) as evidenced by the fact that Carl Doerr[6] finds only 74 cases in the literature. The 62 cases recorded up to 1898 were collected by Schlesinger in that year. There are two principal forms of tuberculosis of the spinal cord proper, namely:

1. Solitary tubercle, better designated conglomerate tubercle. This may arise in two ways, either by hematogenous infection, or by secondary infection by way of the lymphatics from the meninges.

2. Tuberculous myelitis. This is really a meningomyelitis in the great majority of cases, but a few cases of nodular tuberculosis without involvement of the meninges have been described, the first one by Raymond in 1886. The first description of tubercle of the cord was by Wittfeld in 1827, and up to 1870, all cases reported were instances of solitary tubercle. In that year Lionville described tuberculous myelitis. The most extensive publication is that by Schlesinger[7] on "Central Tuberculosis" of the cord.

Doerr reports two new cases: Case 1 is an instance of conglomerate tubercle of the cervical cord in a boy 12 years old. The maternal grandfather died of phthisis and one brother had suffered from tuberculosis of the knee for three years. The patient had been in

---

(6) Archiv. f. Psychiat., Vol. 49, p. 406.
(7) Deutsche Zeitschr. f. Nervenheilk., Vol. 8.

good health until four weeks before death when he was taken with severe headache, diarrhea and irregular fever. Two weeks later he became stuporous and vomited frequently. When admitted to the hospital one week before death he was stuporous, did not respond to questions but groaned with pain. Kernig's sign was present. The neck was slightly stiff but there was no tenderness or deformity of the spine. The left pupil was wider than the right and reacted only slightly to light.

There was slight nystagmus and slight left ptosis. There was an indistinct Babinski sign; the abdominal reflexes and the knee-jerks were present, and there was no ankle-clonus. Ophthalmoscopic examination was negative. The clinical diagnosis was tuberculous meningitis. The anatomic diagnosis was: Tuberculous cerebrospinal meningitis; conglomerate tubercle in the cervical cord; multiple recent and caseous conglomerate tubercles of the brain; miliary tuberculosis of lungs, liver, kidneys and spleen; tuberculosis of the peribronchial glands. It was noted that the cervical portion of the cord was harder than the rest and on section a pea-sized pale red nodule was found to occupy most of the gray matter and much of the adjacent white matter of the right side of the cord. (Plate XII, A.)

Aside from miliary tubercles in the membranes there were no gross lesions in the rest of the cord. The left frontal, occipital and temporal lobes each contained one pea-sized caseous nodule, and in the right optic thalamus there was one of cherry-stone size.

Case 2. A previously healthy man 22 years of age was taken ill with pain and swelling in the right knee which improved on antirheumatic treatment, but some weakness in the leg remained. A year later he was taken with severe pain in the right leg, accompanied by muscular cramps. Later he also had pain in the back, cramps appeared in the left leg, and incontinence of urine and feces set in. At an examination made two months before his death, he was found to be well built, well nourished, and clear mentally. There was no stiffness of the neck, and the eyes were normal. The spinous processes from the third to the seventh dorsal vertebra were tender. The strength in the right arm was considerably diminished, and the circumference of the right arm was three inches less than that of the left. Both legs were completely paralyzed and very atrophic. The knee, ankle, and abdominal reflexes were absent. Pain and touch sense were diminished in the right arm and right shoulder as well as in the entire right leg, but were only slightly reduced in the left leg. The temperature sense was lost on the extensor surfaces of the right thigh and leg but was normal in the left leg. The sense of position was reduced in both legs. Later the strength in the arms became diminished, then the pain became more severe, the patient began to expectorate and developed bed-sores and cystitis.

The clinical diagnosis was transverse myelitis. The anatomic diagnosis was: Conglomerate tubercles in the upper part of the spinal cord and myelitis above and below; chronic tuberculous cerebrospinal meningitis; tuberculous peribronchitis; caseous pneumonia in the upper lobes of both lungs; pericarditis; enlarged spleen; tuberculosis of the right knee; colloid goitre; hemorrhagic erosion of the duodenum; passive hyperemia of the liver and kidneys. The great extent of the tuberculous process on the transverse section of the cord is seen in Plate XII, B. Histologic examination shows typical tuberculous giant-cells and caseation.

TABES DORSALIS.

The Visual Fields in the Optic Atrophy of Tabes. K. Langenbeck,[8] connected with Uhthoff's clinic in Breslau, has had the unique opportunity to study the visual fields in 130 cases of tabes. His conclusions are:

1. There is no form of visual field absolutely characteristic of tabes.
2. Most frequently we find peripheral defect, narrowing of the color fields, and early loss of the appreciation of red and green in the entire field; less frequently partial defect, with well-preserved function in the rest of the field.
3. The rare cases of central defect demand further

---

(8) Klin. Monatsbl. f. Augenheilk., August, 1912.

scrutiny on account of the possibility of retrobulbar complications.

4. Hemianopic fields do not occur in uncomplicated tabetic optic atrophy.

Franke's Operation for Gastric Crises by Avulsion of the Intercostal Nerves. Förster's operation of cutting the posterior roots inside the dura (rhizotomy) was discussed at length in the *Practical Medicine Series,* 1911, Vol. X, p. 166. Franke, another German surgeon, in 1910, suggested the less formidable operation of exposing the intercostal nerves close to the spine and pulling them out, hoping in this manner to injure both the posterior root ganglia and the nerves communicating with the sympathetic ganglia sufficiently to relieve the pain. Sicard and Leblanc[9] have subjected the clinical and experimental data to critical scrutiny, and, to make a long story short, condemn the operation utterly as either useless, if not thoroughly performed, or as absolutely dangerous, if enough of the nerves is pulled out.

LATERAL SCLEROSIS.

Essential or Primary Lateral Sclerosis is discussed by James Hendrie Lloyd and S. D. W. Ludlum.[1] This disease consists in a primary degeneration of the lateral or pyramidal motor tracts. It is not the result of any focal lesion and is not associated with any lesion of other systems such as the sensory columns or the anterior horns. The authors report two clinical cases in which the symptoms suggested this disease but both gave a positive Wassermann test. In a third case necropsy was obtained, and in addition to degeneration of the pyramidal tracts up to the decussation of the pyramids, there was a slight lymphocytic infiltration in the pia of the cord strongly suggestive of syphilis.

In the discussion of this paper Spiller expressed himself in favor of at least a frequent syphilitic etiology both for this disease and the closely related amyotrophic lateral sclerosis and progressive muscular atrophy.

---

(9) Revue Neurolog., Aug. 15, 1912.
(1) Jour. Amer. Med. Assoc., Sept. 28, 1912.

MULTIPLE SCLEROSIS.

Clinical Types. Oppenheim[2] states that multiple sclerosis, because of its frequency, takes first place among the organic diseases of the nervous system and is more than any other likely to be misunderstood and confounded with other nervous diseases. The clinical picture is so varied that it is natural that attempts are made to create different groups, but this is difficult on account of the irregular grouping of the symptoms. However, there are cases in which the symptom-complex remains for a long time either spinal or cerebral while in the classical forms the disease is cerebrospinal.

The spinal forms may be subdivided into the following: Dorsal, cervical, lumbo-sacral and sacral, and mixed.

The *cervical form* has been comparatively recently fully appreciated and Oppenheim therefore sketches it in some detail as follows: "The hitherto healthy and youthful individual falls suddenly ill without apparent cause. The patient has sensations of numbness and awkwardness in one arm, sometimes, but rarely, in both, but when both are affected then one is always affected more than the other. Pains may be an associated symptom. These disturbances increase rapidly, reaching the greatest intensity within days, weeks, or months. The patient complains that he lets objects slip from his hand, that he finds himself unable to button his clothes or to distinguish between his purse and knife in his pocket, etc.

"Physical examination shows the following conditions: The principal symptom is ataxia in one or both arms, and is nearly always a combination of static with locomotor ataxia, accompanied by anesthesia in the hand and the fingers. The sense of posture is the most seriously affected and the most constant symptom; frequently, too, the sensation of touch is involved, and in consequence follows astereognosis, while the sensation of pain and temperature is very often undisturbed. The tendon reflexes of the arms, especially the supinator phenomenon, cannot usually be elicited.

---

(2) Brit. Med. Jour., Sept. 30, 1911.

"A most remarkable fact is that in this cervical form of multiple sclerosis the posterior column symptoms are the most prominent features. I may assert from my own experience that the acute ataxia brachialis is nearly always the result of multiple sclerosis. Yet it is but natural that symptoms often supervene which point towards an involvement of other regions of the transverse section of the cervical cord. These symptoms nearly always lie in the background. For instance, I found in several of my cases a slight atrophy of the small muscles of the hand without change of the electrical excitability or with partial reaction of degeneration. The involvement of the motor tract can be demonstrated by spastic phenomena in one or both legs; Brown-Séquard's syndrome may also be present in some cases, but only indistinctly. However, the symptom-complex is always dominated by the sensory ataxia in the arms.

"The progress is also characteristic. All of our observations show that after weeks and months it comes to a considerable remission; it is even the rule that the symptoms disappear more or less completely, so that the less experienced observer is likely to diagnose curable myelitis cervicalis or a syphilitic affection of the cervical cord. But after a year, or even after a period of years, the disease becomes active once more, or new symptoms indicate clearly the nature of the process. Statements made by Finkelburg go to prove that the syndrome may develop also gradually. This cervical type of multiple sclerosis can exist entirely free from any admixture, but, however, there are cases in which the typical picture is complicated by symptoms appearing at the same time or preceding the attack, which prove the disseminating character of the disease. Thus in some of our cases we found bulbar symptoms together with the cervical; in others an affection of the optic nerve had either developed before the foregoing symptoms or existed at the same time or developed soon after."

The *sacral form* is much rarer than the cervical form and disturbances of the rectal and vesical sphincters are here the prominent symptoms. We may have the ordinary picture of a conus lesion with saddle anesthesia and loss of the ankle-jerk and anal reflex. "Now, in what way is this conus affection characterized as multiple sclerosis? On the one hand, by the fact that the course is, as a rule, not a stable one, not gradually progressive, but runs with remissions. On the other hand, that the vesico-genital symptom-complex is associated with symptoms of disturbances in other parts of the nervous system—for instance, Babinski's reflex or cerebral ocular symptoms."

The complexity of the subject becomes impressed upon us when the author finds that so many spinal cases fail to fit in the classification just given that it is necessary to establish the seven following additional types based upon the part of the transverse section of the cord affected: (1) The typical spastic forms with preponderating localization of the process in the pyramidal tracts; (2) the not infrequent spastic ataxic form which corresponds more or less to the type of combined degeneration of the posterior and lateral tract; (3) the ataxic or posterior tract type, a variety of which—the cervical one—we have already taken into consideration; while another—the rarer one—is sclerotic pseudo-tabes. Now follow the uncommon forms, namely, (4) the type corresponding to poliomyelitis; (5) the syringomyelitis type caused by a diffuse involvement of the gray matter; and (6) the form corresponding to the type of amyotrophic lateral sclerosis. Finally, we still have a (7) type in which the symptomatology has the features of myelitis transversa.

The Cerebral Types. Here the following principal forms can be differentiated:

1. The cerebral form in a restricted sense, in which the symptomatology indicates a localization of foci in the cerebrum.
2. The basal type, in which, according to the clinical symptoms, the central ganglia are the most affected.
3. The well-characterized ponto-bulbar form.
4. The cerebellar form.

In the first and second group hemiplegia is generally the principal symptom and the hemiplegic type of multiple sclerosis was known to Charcot. Apoplectiform seizures are fairly frequent, and there may be aphasia or cortical epilepsy. There are also cases in which mental symptoms are marked.

In some cases the disease for years appears to be confined to the optic nerves, such as the following case related by the author:

"A young woman, unmarried, 34 years of age, with hereditary disposition, always in good health, fell suddenly ill on March 22, 1895, with visual disorder of the left eye—she complained of a mist before the eye. The condition of the fundus was normal, but central acuity of vision diminished rapidly within a few days. The eyeball proved sensitive to pressure. The oculist diagnosed retrobulbar optic neuritis. Rapid improvement was effected by means of diaphoresis and sodic salicylate. Good health followed till June 6, 1897. Then the right eye was attacked in the same way. There was dimness of vision, pain in looking to the right and from pressure on the bulbus; a paracentral scotoma; opthalmoscopic examination was normal; rapid recovery was brought about by means of diaphoresis and sodic salicylate. In September, 1897, the left eye was again affected in the same way—recovering quickly. In 1902, there was temporary diplopia. In the beginning of 1910 the oculist found a marked optic atrophy, a diminution of the acuity of vision, paresis of the right trochlearis associated with paresis of the left. Even after this, improvement and disappearance of the diplopia followed. It was only in the course of these last two years that other symptoms developed, especially weakness of the legs, unsteadiness of gait, and tremor. At an examination in March of this year I found a fully developed sclerosis—namely, spastic paraparesis, absence of abdominal reflex, intentional tremor, and partial optic atrophy."

TRAUMATIC AFFECTIONS OF THE CORD.

The Traumatic Diseases of the Spinal Cord With Report of a Case of Progressive Muscular Atrophy from Overwork. This subject is discussed by Erb (8) who first calls attention to his report fifteen years ago of two cases which led him to believe that progressive spinal muscular atrophy occasionally is caused by slight accidents and muscular fatigue.

---

(8) Deutsche Zeitschr. f. Nervenheilk., Vol. 45, p. 1.

[This matter naturally is of the greatest medico-legal importance and was discussed in the *Practical Medicine Series,* 1911, Vol. 10, p. 130, in connection with a report by Astwazaturow of a case ascribed to a fall. An occasional traumatic origin has also been claimed with considerable plausibility for the closely related disease, amyotrophic lateral sclerosis.—ED.]

Erb's new case is that of a viticulturist 31 years old whose previous history was entirely negative. On April 7, 1910, after cutting bunches of grapes for two hours he attempted to cut an unusually thick stem, which required a powerful muscular effort, and suddenly experienced severe pain in the ball of the right thumb, particularly in the muscles located between the metacarpal bones of the thumb and the index finger. The pain remained, but there was no swelling, no ecchymosis and no paralysis. He began to use the left hand for his work and in the course of two weeks the pain disappeared. Two or three months later he noted that the right hand gradually grew weaker, that the small muscles of the hand were wasting, and that the same was true to a lesser extent of the musculature of the entire right upper extremity. There was no sensory disturbance of any kind.

Examination of the upper extremity showed the typical picture of progressive spinal muscular atrophy, with advanced atrophy of all the small hand muscles. The latter gave a complete reaction of degeneration. In the rest of the extremity the atrophy was of more moderate extent and the change in the electric reaction incomplete. Fibrillary twitchings were present. The muscles of the left upper extremity were entirely normal. The tendon reflexes were decidedly increased in both arms and legs, and in the latter more so on the right side. There was a suggestion of ankle-clonus but no Babinski sign. When examined a month later there were occasional fibrillary twitchings in the left arm, and the reflexes were more increased, with prolonged ankle-clonus but no Babinski sign.

Erb considers this a text-book case of progressive spinal muscular atrophy and not one of amyotrophic lateral sclerosis because of the absence of spasticity of the muscles and of the Babinski sign. At any rate the differentiation between these two diseases is unimportant as they are closely related and frequently overlap. Erb believes that the disease in this case was produced by two factors, namely, the habitual overwork of the muscles of the right hand, and the trauma affecting these same muscles. Of the two factors the former is considered the more important. In this connection he discusses Edinger's exhaustion theory, the extensive application of which by Auerbach was discussed in the *Practical Medicine Series,* 1911, Vol. 10, p. 14; and while Erb does not agree with those authors in every detail, he believes that the principle holds for the disease in question.

Being called upon to give expert testimony in this case, Erb testified that in his opinion the disease was brought on by the injury mentioned, and his opinion was sustained by the court. Erb has collected all available cases in the literature which have a bearing on the problem at hand, and includes both progressive spinal muscular atrophy and amyotrophic lateral sclerosis. On the other hand he does not consider that any evidence exists in favor of a traumatic etiology for muscular dystrophy (idiopathic muscular atrophy).

The cases are divided into three groups:

1. Cases of pure injury (commotio) of moderate degree without severe or permanent ordinary traumatic complications.
2. Cases of pure muscular overwork.
3. Cases in which both trauma and overwork took part.

In the first group there are 16 cases. Among these is one previously reported by Erb.[4] A Russian government official with negative previous history sustained two moderately severe injuries within four weeks, namely, a fall on the back and a blow at the back of the neck. A month later atrophy commenced in the right arm, then in the left arm, back and legs, finally

---

(4) Deutsche Zeitschr. f. Nervenheilk., Vol. 38, p. 61.

presenting the typical picture of progressive spinal muscular atrophy.

Another case which Erb considers of great importance is that of Giese.[5] A machinist, 39 years old, sustained a severe psychic trauma through the breaking of a fly-wheel in his shop, and by his presence of mind he prevented severe injury to all those employed. He was not physically injured but was unable to speak for several hours. The next day he appeared fully recovered, but during the next few months he showed some change and often appeared tired. Six months later he fell ten feet, landing on his feet, then falling with his right elbow on an iron plate which caused a slight contusion above the elbow. A few weeks later there developed weakness in the right hand and arm and pain and paresthesias in the right forearm. Four months after the accident there was distinct atrophy of the small hand muscles. The reflexes in the arms were normal, those in the legs were increased. A few months later the clinical picture had become that of typical amyotrophic lateral sclerosis, with bulbar involvement, greatly increased reflexes, knee- and ankle-clonus, fibrillary twitchings and with normal sensation. Erb's explanation is that the psychic trauma acted as a predisposing cause, while the disease originated from the "commotio" produced by the fall, and the slight injury at the elbow merely served to determine the primary localization of the symptoms. This is a mechanism long recognized, first concussion with molecular changes, then degenerative and progressive changes.

In the second group, that of pure overwork without any gross injury, there are very few cases which will bear close scrutiny. Here belongs the case related at the beginning of the article, as Erb states that the more the thinks the case over the more he becomes convinced that the overwork of the right arm was the only important factor. The only other case which he accepts as due to muscular fatigue is that of Ottendorff.[6] A male servant, 28 years old, was working on a ladder which began to give way and

---

(5) Deutsche med. Wochenschr., 1904, p. 1348.
(6) Monatschr. f. Unfallheilk, 1902, Vol. 9, p. 318.

he had to hold on to a post with his arms for several minutes during which he was in a state of great fear, until some one readjusted the ladder. This was in August, 1900, and he showed no symptoms until a few weeks later when he complained of pain, paresthesias, stiffness and weakness in the arms, especially in the right one. In March, 1901, atrophy of the interossei and increased tendon reflexes both in the arms and legs were noted. By May, 1902, he had developed the full clinical picture of amyotrophic lateral sclerosis with bulbar involvement. Erb considers the chief etiologic factor the muscular exertion on the one occasion.

In the third group, trauma and over-exertion, are the two cases reported by Erb in 1897, and one by E. Meyer.[7]

INTERMITTENT CLAUDICATION.

Intermittent Claudication of the Spinal Cord is the subject of a lucid clinical lecture by Déjérine[8] who described this affection many years ago. He states that it is usually the forerunner of spastic paralysis of the legs, and if recognized in time it is possible to institute treatment which may prevent the further evolution of the cord disease. He described the case of a woman, 37 years old. She noticed that in walking the left leg would tire easily and feel heavy so she frequently had to sit down and rest. A few moments' rest would relieve her completely but the trouble would soon return. Three years later the same symptoms appeared in the right leg. Gradually the time which she could walk continuously lessened so much that finally she could walk only 600 feet between pauses.

On examination no motor weakness was found and no sensory disturbance. The tendon reflexes were increased, more so on the left side. Babinski's sign was present on both sides. In this case the condition of the reflexes remained as stated also during rest, but in certain cases of this kind the Babinski sign appears only during the state of fatigue. This patient also presented intermittent sphincteric disturbance in the form

---

(7) Münch. med. Wochenschr., 1901, p. 172.

(8) Presse Méd., Nov. 29, 1911.

of retention. In this case there was no sign of intermittent claudication of the extremities themselves. The pulsation in the vessels of the legs was normal and there was no coldness or cyanosis. The existence of changes in the reflexes and the sphincteric affection indicate a disturbance of the cord which is best explained by an intermittent constriction of the arteries in the lumbar region of the cord.

It is now twenty years since Déjérine first encountered this symptom-complex. Men are more frequently affected than women, just as in the peripheral form of intermittent claudication. The onset is usually in one leg but sooner or later, sometimes not until some years have elapsed, the other leg becomes involved and gradually the picture of a spastic paraplegia develops. Sphincteric disturbances also usually develop, such as incontinence and precipitate urination. The tendon reflexes are always increased and generally more so on the side most affected.

As to etiology, the condition nearly always is an early symptom of slowly developing syphilitic paraplegia. In fact, this is the only cause of practical importance, and the only other infection which may be a causative factor is influenza. In the patient whose case was just related, the Wassermann test was positive.

As to intermittent claudication of the legs due to affection of the peripheral arteries Déjérine states that it was first described in horses by Boulay in 1831 and in man by Charcot in 1858. In the horse, too, it is true that there is no pulsation in the affected leg during the attack, and at autopsy one usually finds an obliterating endarteritis near the bifurcation of the abdominal aorta, generally in the iliac arteries. The cause of the arteritis is generally the invasion of the intima by the larva of a round-worm, the *Sclerostoma equinum.* Déjérine is very skeptical concerning the influence of tobacco in intermittent claudication.

The Lumbar Type of Intermittent Claudication. Since Charcot first described this characteristic vascular disturbance in 1858, a great many clinical types have become recognized and derive their characteristics from the vascular area involved such as those supplied by the tibial, brachial, coronary, mesenteric, cerebral and spinal arteries. Now J. Ramsey Hunt[1] describes a *lumbar type* of intermittent claudication. He reports a case of a stockbroker, 51 years old, who had indulged excessively in alcoholic beverages and in rich food but had not used tobacco for twenty years. He had syphilis twenty years ago and a severe attack of articular rheumatism fifteen years ago. Two years ago he began to have attacks of pain in the lower part of the back but only when walking. After standing a few minutes and leaning against a fence or lamp-post the pain would disappear. While sitting, lying in bed or on first rising in the morning there was not a trace of pain or stiffness in the back. The pain was severe, and if he attempted to continue walking after its appearance, it would develop into a painful cramp-like feeling in the small of the back which would bring him to an abrupt standstill. After a brief rest with support, the pain and cramp would always promptly disappear. The pain was fairly well localized in the lower lumbar region, and when very severe, would radiate somewhat into the sides but never as far forward as the abdomen.

On examination it was found that movements of the spinal column were perfectly free in all directions, the pupils and tendon reflexes were normal; in fact the entire neurologic examination was negative except for slight tremor of the hands. The heart-sounds were normal, the pulse of moderate tension and the urine was normal. Hunt believes that the symptoms in this case were due to disease of one or more of the lumbar arteries or to an arteriosclerotic process in the abdominal aorta, which interfered with the free flow of blood through these vessels, so that a sufficient blood-supply was received for the physiologic processes in the passive state, but not for the increased demands of the sacro-lumbar mass during activity. Hence the painful cramp and its peculiar intermittent character.

SPINAL CORD DECOMPRESSION.

Spinal Decompression. The benefit derived from

---

(1) Amer. Jour. Med. Sciences, February, 1912.

decompression operations on the head in cases of brain tumor has now become recognized everywhere but it is only recently that experiences have accumulated which tend to show that symptoms of spinal cord disease may be similarly relieved. Pierce Bailey and Charles A. Elsberg[3] report 7 cases in which the results are summarized as follows:

"The improvement in most of the cases was very striking, although in two the changes in symptoms which followed the operation could not be construed as benefits to the patient. In one patient, severe pain of three years' duration was promptly relieved; in another there was almost complete recovery from the symptoms of a lesion at the level of the eighth dorsal segment, an atypical Brown-Séquard syndrome with unilateral sensory loss and spastic motor palsy of both lower limbs; in a third, disappearance of an unilateral sensory and spastic motor paralysis of one leg. In one patient only temporary changes in the symptoms occurred; these were of physiologic interest, but without practical benefit to the patient. In a case of infiltrating tumor of the cauda equina the pain disappeared and the anesthesia grew less; in a case of intramedullary tumor the patient, bedridden before the operation, regained the power of walking without assistance; in still another, the anesthesia grew less.

"We have arranged our cases in three classes: (1) those in which nothing pathologic was found within the dura and in which the operation was followed by the complete disappearance of all the symptoms; (2) cases in which nothing was found at operation, which was followed by temporary marked improvement; and (3) cases in which a tumor was found which could not be, or could only partially be, removed.

"We are fully aware that in the last group of patients the improvement which followed the primary operation was actually due to the decrease in the intradural pressure after the free removal of spinous processes and laminæ and incision of the dura. These are instances of true spinal decompression, and we have included them because, in direct contrast to the other cases, they demonstrate the benefits that may follow a laminectomy when the intradural pressure is markedly increased. But in the five other cases there was no evidence of an increase of intradural pressure. In these five patients the improvement observed—both in symptoms of functional character such as pain, and in organic symptoms such as paralyses, anesthesias, etc.—leaves no room for doubt that surgical exposure of the spinal cord, even in the absence of increased intradural pressure, may effect some change which temporarily or permanently benefits or checks the symptoms of local spinal cord disease.

---

(3) Jour. Amer. Med. Assoc., Mar. 9, 1912.

"We are not in a position to offer a physiologic explanation of the phenomenon, although it is probably connected with the circulation in the cord itself. The infrequency of necropsies in these cases throws the burden of explanation on experimental work. In Allen's experiments[4] on spinal injuries in dogs, drainage of the spinal cord seemed the condition necessary for recovery. Before Allen, Horsley had expressed the view that 'probably many cases of so-called acute myelitis are really meningeal in origin, and that a laminectomy and free drainage of the subdural space might arrest the whole process and the subsequent fatal injury which the cord sustains in such cases.'

"It may be that in our cases, the operation of laminectomy and incision of the dura effected vital changes in the blood-supply of the cord, or exerted a beneficial influence by the admission of light and air, analogous to the effect of laparotomy in tuberculous peritonitis. In most of our cases the operation went no further than an exploratory laminectomy with opening of the dura, and we have ascribed the resulting changes in the symptoms to the effects of spinal decompression. We have made use of this term 'spinal decompression' as a convenient means of expression, realizing fully that in five of our cases the results from the operations were not entirely analogous to the results of cerebral decompression.

"Whatever the explanation may be, the facts struck us as worthy of attention, and we began to inquire if

---

(4) Jour. Amer. Med. Assoc., Sept. 9, 1911, p. 878.

such effects had not been recorded before. We soon found, scattered throughout the literature on spinal cord tumor, reports of cases in which relief of symptoms, notably pain, followed operations undertaken for spinal cord tumor, when no tumor was removed. The most suggestive paper on this subject is a clinical lecture by Sir Victor Horsley[5] entitled 'Chronic Spinal Meningitis.' By this he understands an affection of the spinal meninges with some sclerosis and wasting of the cord, usually, though not always, syphilitic in character. The symptoms are those of a progressive sensorimotor paralysis, practically indistinguishable from the symptoms of spinal cord tumor.''

[The subject of ''localized spinal serous meningitis'' has been discussed in previous volumes of the *Practical Medicine Series* (particularly 1909, Vol. X, p. 165)]. In the following cases complete relief was obtained:

Case 1. *Paraplegia dolorosa of three years' duration. Osteochondritis of the twelfth dorsal vertebra. Operation, recovery.* A man 40 years old had suffered for three years from attacks of pain in the lower part of the back and from pain radiating down the legs. There was marked rigidity of the dorsal and lumbar regions of the spine with tenderness to pressure, most marked over the second lumbar vertebra. There was no paralysis or sphincter disturbance. The right knee-jerk was absent, the left one diminished. Sensation was impaired from the level of the distribution of the twelfth thoracic segment down. On laminectomy the laminæ of the twelfth thoracic vertebra were found thickened, soft and grayish. The pathologic diagnosis was chronic osteochondritis with new bone formation. The dura was slightly congested and the cord appeared normal. The relief from pain was immediate and the anesthesia disappeared in a few weeks. Both knee-jerks were absent for a time but were present and equal six weeks after the operation. Eight months afterwards the patient was in perfect health.

Case 2. *Suspected tumor of the eighth thoracic segment. Laminectomy with negative findings. Complete recovery.* A man 40 years old had suffered from pain

---

(5) Brit. Med. Jour., 1909, i, 513.

in the middle of the back and weakness in the right leg for six months. He had occasional shooting pains in the legs and a girdle sensation around the middle of the body. The sexual power was lost. After three months the left leg also became weak and there was difficulty in urination. Both lower extremities were spastic, with increased reflexes, and the Babinski sign was present on the left side. Sensation was diminished in both lower extremities. At the operation the arches of the fifth, sixth, seventh and eighth thoracic vertebræ were removed, and the dura opened, but no pathologic condition was discovered. Nevertheless the result was remarkable. Eighteen days after the operation the patient walked with a cane and most of the spasticity had disappeared after four weeks. The sensory disturbances disappeared, sexual power returned, and the patient went back to work. When last heard from one year after the operation, he was perfectly well.

Case 3. *Suspected tumor of the spinal cord. Laminectomy with negative findings. Complete disappearance of all spinal symptoms.* A man 30 years old gave a history of an attack about ten years ago of a severe pain in the back lasting eight days. Six years ago the pain returned and from that time on he had similar attacks every few months, with increasing severity. The right abdominal reflexes were not obtained. The reflexes in the left leg were increased, with ankle-clonus and the Babinski sign. The left leg was weaker than the right and there was marked hyperesthesia in the distribution of the twelfth thoracic and first lumbar roots. Sensation was diminished in the distribution of the third and fourth lumbar roots. The Wassermann test and *x*-ray examination were negative. The arches from the ninth thoracic to the second lumbar vertebræ were removed, but the findings were entirely negative. The pain disappeared, almost at once, and three weeks after the operation all of the sensory disturbance had disappeared and one month after the operation the pathologic reflexes were no longer present.

DISEASES OF THE PERIPHERAL NERVES.

THE CRANIAL NERVES.

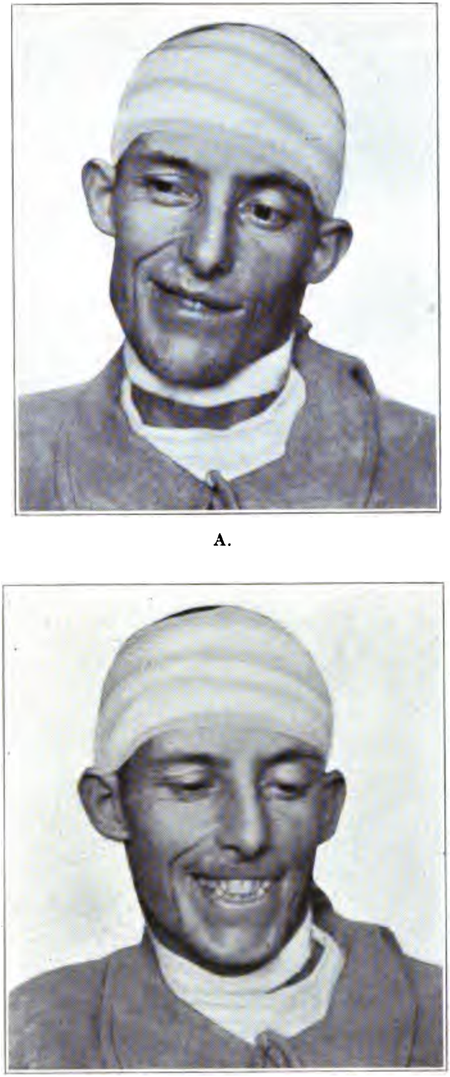
The Etiology of So-Called Rheumatic Facial Paralysis. Jendrassik[6] believes that in the ordinary refrigeration or "rheumatic" facial paralysis we are not dealing with a neuritis but with a nerve degeneration due to compression caused by periostitis of the Fallopian canal. It is striking that the facial nerve is the only nerve which becomes diseased from exposure. The author does not believe that exposure to cold or draught in itself can cause the paralysis as the whole nerve is affected and the trunk is well protected both in the bony canal and in the substance of the parotid gland. He believes that infection reaches the bony canal from the middle ear or through the lymphatics from the mouth. The inflammation probably does not extend to the nerve. A further proof of the correctness of this assumption lies in the fact that at the onset the patient usually complains of pain and tenderness located in the temporal bone. The fact that recovery is the rule also speaks against the existence of an actual neuritis, but as the periostitis of the bony canal is merely a simple inflammation it subsides without causing permanent compression.

The same subject is discussed by Karl v. Ketly.[7] The paralysis rarely appears until twelve to twenty-four hours after exposure to cold, and it then affects the entire nerve at once and does not extend successively from one branch to another. There have been very few opportunities to study the pathologic anatomy of these cases. In 1891 Minkowski reported the case of a patient who died of accidental poisoning seven weeks after the onset of facial paralysis following exposure to cold. The nerve in the Fallopian canal and peripherally to it showed marked degeneration but there was no sign of inflammation in the neurilemma. However,

---

(6) Neurol. Centralbl., June 16, 1912.
(7) Deutsch. Arch. f. klin. Med., May, 1912.

PLATE XIII.

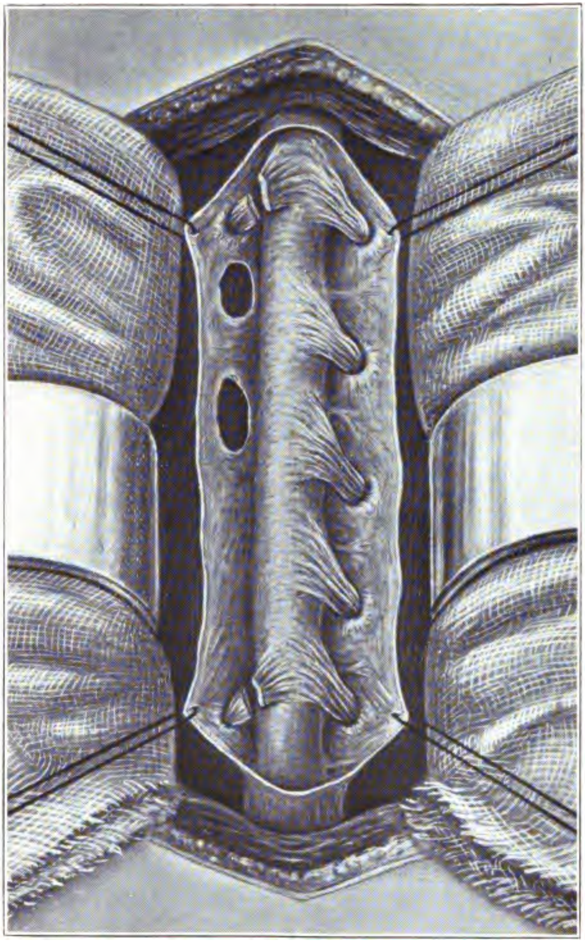


A.

B.

Partial paralysis of facial nerve with loss of emotional movement.
A. In smiling only right side of face contracts.
B. In showing teeth both sides contract.—W. G. Spiller. (Page 155.)

PLATE XIV.



Avulsion of left sixth, seventh and eighth cervical nerve roots. Fifth cervical and first thoracic posterior roots severed at the operation.—Frazier and Skillern. (Page 159.)

it cannot be excluded that an inflammation had existed which caused the nerve degeneration, and then entirely subsided.

As to the way in which exposure causes the paralysis, there are various opinions. Most authors follow Erb who says that the inflammation caused by the cold attacks the nerve at the outer opening of the Fallopian canal and from there may extend as far as the geniculate ganglion. Against this view the author states some of the points already mentioned, namely the protected position of the nerve beyond the canal, the failure of individual branches to be affected alone and the practical absence of cases of bilateral facial paralysis, although it would appear common enough for both sides of the face to be exposed to cold or draught. The frequency with which the branches to the stapedius and the chorda tympani are affected early is also proof that the cause of the paralysis must be sought high up in the Fallopian canal.

In 1904 Strenger pointed out that the facial nerve was most readily affected by cold while in the wall of the middle ear, and he believes that the cold air entering through the Eustachian tube causes the trouble. In two cases he found the opening of the Eustachian tube abnormally wide. Ketly rejects Strenger's theory because the cold air becomes warmed in the mouth, and after passing through the long and narrow tubes it is no longer capable of causing paralysis by affecting the nerve in the wall of the middle ear. And if this theory were correct we should frequently find bilateral cases as there are two Eustachian tubes.

Von Ketly believes that chilling can come only by way of the external auditory meatus. In this roomy canal it meets with only one resistance, namely the thin tympanic membrane. In rare cases there is also involvement of the auditory nerve and as this can only be affected by way of the tympanum, we have here another reason for believing that the refrigeration takes place by way of the external canal.

Peculiar Behavior of Electric Reactions in Peripheral Facial Paralysis. Toby Cohn and Emma Gatz-

Emanuel[8] report two cases which show that there are exceptions to the ordinary rules of parallelism between the degree of functional disability and the reaction of degeneration. On the one hand there are cases in which normal function is almost fully restored and yet the muscles remain unresponsive to the farradic current, while in others the reverse is the case, the electric response becoming almost normal while paralysis persists.

The first case is that of a female 20 years old who had developed right-sided facial paralysis following an attack of measles at three years. All of the muscles supplied by both the upper and lower divisions of the right facial nerve were completely paralyzed but only the muscles supplied by the upper division, namely the occipito-frontalis and the orbicularis palpebrarum, showed lack of electric response. The other muscles reacted practically normally to both currents.

Similar cases have been reported by others and explained in various ways. Thus, Placzek in 1893 reported three cases of facial and one of brachial paralysis, of long standing, with normal electric reactions and offered the explanation that only the axis-cylinders of the fibers were affected. In 1905 Sossinka, a pupil of Cohn's, recorded a similar case in a woman 25 years old whose left facial nerve had been paralyzed after an injury to the mastoid bone at the age of three years. As the electric reactions were normal the author concluded that the loss of power was of a functional nature, due to the loss of the idea of motion caused by prolonged inactivity and the lack of peripheral production of sensation of muscular movement, from a complete lack of treatment, which was undertaken only at the age of 25 years.

Cohn explains the failure of return of muscular power in these cases on the same grounds as we explain the failure of deaf children to acquire speech. Those patients who developed their facial paralysis in early childhood and never had muscular contractions produced in the only way possible, namely, by electric stimulation, had no occasion to develop the necessary

---

(8) Neurolog. Centralbl., Feb. 1, 1912.

psycho-motor mechanism in connection with the affected muscles and therefore could not originate any function, although regeneration had occurred. The correctness of this view was demonstrated by the fact that electric treatment in Cohn's case soon caused a return of muscular power. In his text-book on electrotherapy, Cohn proposes the term "metaparalytic psychogenic akinesia" for this type of muscular disability and wishes it distinctly understood that while the condition is psychogenic it is not, properly speaking, hysterical.

A somewhat similar explanation has been offered by Ehret (9) for certain cases of traumatic paralysis of the peroneal muscles which he calls "habit" paralysis, caused by prolonged pain which makes the patient keep the muscles at rest for such a long time that he seems to have, in a sense, forgotten how to use them after the disappearance of the pain.

The second case reported by Toby Cohn and Emma Gatz-Emanuel is one of a woman who in 1907 had a sarcoma removed from the right parotid gland and immediately afterward developed facial paralysis. When examined six months later she had complete facial paralysis with complete reaction of degeneration. Three months later some of the cheek muscles began to react a little to the faradic current. Electric treatment was continued for four years without the slightest improvement in the use of the muscles, but the electric conditions gradually improved so as to approach a normal condition in some of the muscles. Ordinarily the return of electric response warrants a good prognosis but in this case there was no functional improvement whatever. The authors are unable to explain the condition in this case.

Loss of Emotional Movement of the Face with Preservation or Slight Involvement of Voluntary Movement in Partial Paralysis of the Facial Nerve. W. G. Spiller (1) calls attention to the fact that emotional movement of one side of the face may be lost while voluntary movement may be preserved or only slightly impaired. He has observed this phenomenon, especially in a case of slight facial weakness from pressure of a tumor in the cerebello-pontine angle upon the facial nerve. This patient contracted only the muscles of the right side in smiling (Plate XIII) but in showing the teeth there was little impairment on the left side. The tumor was located and the operation performed by C. H. Frazier.

---

(9) Archiv f. Unfallheilk, II, p. 32.
(1) Amer. Jour. Med. Sciences, March, 1912.

Bechterew believes that the emotional movements in man are accomplished by means of special tracts which have no relation to the tracts concerned with voluntary movement. Nothnagel makes the statement that when the voluntary movement of the face is lost in hemiplegia from a facial lesion, but both sides of the face take part in equal degree in emotional movement, one may assume that the optic thalamus and its connections with the cortex are uninjured. Von Monakow also believes that a part of the sensory reflex tracts for laughing or crying is situated in the optic thalamus, but the work of Roussy and Ernest Sachs throws doubt upon this theory.

Pupillary Disturbances in Chronic Alcoholism are discussed by Nonne[2] who has had his attention focused on this subject for years. In 1908 he reported 18 cases of loss of the pupillary light reflex among 1,460 severe cases of chronic alcoholism. Nine of these cases had their spinal fluid examined for cells and globulin with negative results which further tended to exclude syphilis, no history or evidence of which was obtainable. Since then he has had three cases of severe chronic alcoholism without history of syphilis and with negative Wassermann test in which the pupils failed to react to light. That great care must be taken in observing such cases is shown by the experience of Weiler[3] who among 1,000 alcoholics found 8 cases without clinical signs of syphilis, tabes or general paresis, whose pupils did not react to light, but all of them gave positive Wassermann tests, and subsequently two of the patients developed symptoms of tabes. Weiler's material, therefore, naturally led him to the conclusion that syphilis

---

(2) Neurolog. Centralbl., Jan. 2, 1912.
(3) Zeitschr. f. d. ges. Neurol. u. Psych., II, 1910, H. 2.

is an essential factor in the production of the Argyll-Robertson pupil.

Other authors have also reported cases in which the pupillary disturbance existed as the only physical sign for many years before the development of other signs of tabes. In his excellent book on pupillary disturbances in nervous and mental diseases, Bumke takes the stand that while individuals who apparently are only alcoholics, have shown loss of pupil reaction for years without any symptoms of syphilitic or parasyphilitic disease, yet "theoretically we must say that so far no case is on record in which the existence of syphilis can be excluded."

Nonne now believes that he has a case which fulfils the requirements, namely one of severe chronic alcoholism which showed no other sign of organic nervous disease than the Argyll-Robertson pupil, with no psychic symptoms, with all "four reactions" negative, and where neither brain nor cord at necropsy showed anything suspicious of paretic dementia. The patient was a merchant, 51 years old, who had been a heavy drinker. He denied venereal infection, he was poorly nourished, had a dilated heart, emphysema, slight albuminuria, and slight edema of the lower extremities. The pupils were equal, moderately dilated, did not react to light but reacted on convergence. They were repeatedly tested both by daylight and artificial light and always with the same result. The Wassermann test with the blood and spinal fluid and the globulin tests were negative and there was no increase in cells. Mentally, the patient on admittance to the hospital showed some alcoholic excitement, but after its subsidence there was no sign of dementia.

The necropsy revealed a dilated heart, with degenerated heart muscle, but the aorta was normal and entirely free from signs of syphilitic aortitis. The brain was thoroughly studied macroscopially. The pia was slightly edematous, but the convolutions were normal and showed no atrophy and the brain vessels were normal. The cord showed no gross or microscopic changes. Hence all forms of syphilitic and parasyphilitic disease may be excluded.

This case also shows the opinion to be erroneous that the Argyll-Robertson pupil is always associated with some degree of degeneration of the posterior columns of the cord. Nonne declares that, *à priori,* there is no reason why alcoholism *per se* may not produce the Argyll-Robertson pupil. Bumke himself reached the conclusion that the seat of the lesion causing the loss of light reflex probably is at the location where the reflex impulse passes from the sensory to the motor portion of the arc, that is, in the termination of the filaments of the centripetal reflex fiber around the nucleus of the fibers for the sphincter of the iris. We know that in chronic alcoholism there is considerable proliferation of glia and in this case it may have affected the locality mentioned.

[It is rather strange that under these circumstances the brain was not subjected to a minute histologic examination of the condition of the glia, but very likely this is reserved for a future and more complete communication.—Ed.]

Soon afterwards another case came to Nonne's notice. An alcoholic laborer 50 years old, who denied syphilis, came to the hospital on account of fears and mental depression. The only neurologic finding on examination was failure of the pupils to react to light. The Wassermann test on the blood and spinal fluid and the examination of the latter for globulin and cells were negative. Three of the patient's four children were examined and showed no signs of syphilis. The patient's nervous symptoms subsided after six days of abstinence, but the pupils remained unchanged during three weeks of observation.

That the Argyll-Robertson pupil of alcoholics may disappear after a period of abstinence is shown by the following case: A man 44 years old who had been a heavy drinker for ten years, came to the hospital on account of delirium tremens. He showed no evidence of pathologic arteriosclerosis and there were no findings except the loss of pupillary light reaction. The "four reactions" were entirely negative. Two weeks after the cessation of the delirium tremens the pupils began to react sluggishly to light, and four days later the reaction had become entirely normal.

THE SPINAL NERVES.

Case of Brachial Plexus Paralysis with Avulsion of the Anterior and Posterior Spinal Roots. C. H. Frazier and P. G. Skillern[4] report an unique case of avulsion of the 6th, 7th and 8th cervical spinal roots within the dural sac.

The patient, a middle-aged physician, was struck on the head and shoulders by a beam falling from the fourth story of a house. After recovering from the shock of the accident, it became evident that he had sustained an injury to the brachial plexus. There was complete anesthesia for pain, touch, heat and cold, up to the elbow, hyperesthesia for a distance of three inches above the elbow and hyperalgesia in the shoulder, neck and upper chest. There was complete paralysis of all the muscles of the left upper extremity, including the deltoid and the pectoralis major and minor, and some weakness of the sternocleidomastoid. The rhomboidei, levator anguli scapulæ, and the serratus magnus were not involved. The reflexes of the upper extremity were altogether abolished, those of the lower extremity exaggerated. The oculopupillary phenomena, *i. e.*, the narrowing of the palpebral fissures, contraction of the pupil and enophthalmos, indicated an injury to the ciliospinal fibers of the sympathetic. There was some evidence of injury to the seventh cervical and first thoracic vertebræ. The symptom-complex indicated an involvement of structures supplied by the sixth, seventh and eighth cervical nerves, with impairment of function or irritation of the fourth and fifth cervical and the second thoracic. In addition to this, however, there was some hyperesthesia in the distribution of the superficial cervical nerves of the cervical plexus (C_1, C_2, C_3). There was also some functional disturbances of the left lower limb, which, as Spiller suggested, might be produced by congestion of the left anterior horn of the cervical swelling such as might result from an injury to the cells by pulling on their axis-cylinder processes.

"The determination of the precise seat of the lesion,

---

(4) Jour. Amer. Med. Assoc., Dec. 16, 1911.

that is, whether within or without the dural sac, was, however, a matter of the utmost importance in this case. The pupillary phenomena are, when present, absolutely conclusive evidence that there is an intradural lesion at least of the eighth cervical and first thoracic. On the other hand, though the sensory disturbance pointed clearly to the involvement of the fifth, sixth and seventh cervicals, that is, the superior portion of the cervical plexus, the absence of paralysis of the rhomboidei, the levator anguli scapulæ and especially the serratus magnus was equally conclusive that these three nerves could not all have been injured within the dural sac. Even after the character and extent of the lesion were revealed it was not altogether easy to account for the sensory and motor phenomena as elicited before the operation. For example, the presence of anesthesia in the cutaneous supply of the intercostohumeral nerve was somewhat perplexing as was the hyperesthesia in the distribution of the cervical plexus. However, there was evidence in the lower extremity of an injury to the anterior horns of the cord itself and it is quite possible that in the avulsion of the roots the posterior segment suffered also. The discovery with the radiograph of some separation of the seventh cervical and first thoracic vertebræ was at least suggestive of an intraspinal lesion. There were at all events some perplexing features and if we add to these the neurotic and emotional temperament of the patient we increase still further the difficulty of accurate localization.

"For the purpose of relieving the intense neuralgia of which the patient was complaining, we planned to cut the posterior roots of the fifth, sixth, seventh and eighth cervical and first thoracic nerves. Accordingly on May 23, 1910, a laminectomy was performed and when the dural sac was opened, we were astonished to find the following conditions: On the left lateral aspect of the canal corresponding to the level of the sixth cervical and the first thoracic vertebræ there were two defects in the dura each as large as a ten-cent piece, and on the same side both the anterior and posterior roots of the sixth, seventh and eighth cervical nerves were absent. Plate XIV. The roots evidently had been torn completely from the cord. As three of the five roots were absent it remained for the operator only to cut the fifth cervical and first thoracic roots. This was easily accomplished and the wound in the dura, musculo-aponeurotic, and cutaneous structure was closed.''

The patient left the hospital 27 days after the operation. The pain had become more bearable, and the burning sensation in the distribution of the 4th cervical and 2d thoracic nerves had in large measure disappeared. He improved and some months later he was able to resume his practice to a limited extent.

The authors review the literature of the subject and formulate the following conclusions:

1. The case herewith presented is the first on record in which the seat of the lesion within the dural sac was discovered at operation.

2. Avulsion of the plexus, partial or complete, without skeletal injury, is comparatively infrequent. There are recorded all told but twenty-one cases in which the precise nature of the lesion has been vertified by operation.

3. In the mechanism of avulsion traction is the most influential factor.

4. In the pathology emphasis is laid first on the presence and on the injurious effects of the traumatic exudate, the organization of which interferes with nerve regeneration; and secondly, on the possibility of an intraspinal lesion (either avulsion of the roots or degeneration of the cord) associated with an extraspinal lesion, as a clue to the larger percentage of failures following reconstructive operations on the plexus as compared with similar operations on the peripheral nerves.

5. The authors failed to find a single instance of complete restoration of function. Without operative interference the outlook is almost invariably hopeless. Unpardonable delay in operating may account for some failures. Six years may elapse before the end results manifest themselves.

6. In view of the gravity of the lesion, of the result-

ing disability and of the occasional intractable neuralgia, surgeons should insist on immediate resort to operation.

7. If, at the operation in very recent cases, the nerves are found swollen and their continuity not altogether destroyed, an incision should be made into the nerve to allow of the evacuation of the exudate.

8. When the approximation of the ruptured nerve is impossible, a cross anastomosis may be affected with the nerve on the unaffected side, as first proposed by Alexinsky.

9. In cases of intractable neuralgia one should not hesitate to resort to intraspinal section of the sensory roots.

Occupation Neuritis. M. Mirowsky[5] reports several illustrative cases from Strümpell's clinic at Breslau. The cases are divided in four groups.

I. *Cases Dependent on Repeated Direct Trauma to the Nerves During the Work.* Case 1. A lithographer, 56 years old, was constantly working with the right arm strongly flexed and the elbow resting on the table, with the hands flexed and often pronated, and the fingers spread apart. By working in this position there was a constant pull on the ulnar nerve but the latter was not subjected to direct pressure. He began to complain of a tingling in the right forearm accompanied by palpitation of the heart. There was diminished tactile and pain sense in the little and ring fingers and also diminished tactile sense in the ulnar portion of the hand. The ulnar nerve was not tender to pressure and there was no atrophy or disturbance of motility. In the left hand, which was subjected to less strain, the disturbance was much less. Similar ulnar neuritis, associated with atrophy of the ball of the thumb and the small finger muscles, occurring in lithographers, has been described by Vogt.

II. *Cases in which the Worker is Exposed to Poisonous Substances such as Lead, Arsenic and Carbon Bisulphid.* In these cases the habitual use of certain muscle groups to the point of fatigue may be of importance in the localization of the paralysis.

---

(5) Deutsche Zeitschr. f. Nervenheilk, Vol. 45, p. 3[illegible].

Case 2. A man, 51 years old, addicted to the use of alcohol, had worked for fifteen years in zinc works. He developed burning pain in the left foot which increased to such an extent that he had to give up work. First the left and then the right foot began to drag and he finally developed bilateral foot-drop. The knee- and ankle-jerks were very brisk. Sensation was diminished in both feet. There was slight atrophy of the balls of the thumbs but no paralysis in the upper extremities. The paralyzed muscles in the legs showed the reactions of degeneration. The author looks upon this case as a zinc neuritis, and believes that the alcohol at most has been merely a contributing factor.

Case 3. A man 47 years old, a zinc worker, also showed impairment of the muscles supplied by the same nerves as those involved in the previous case, namely, the median, ulnar, peroneal and tibial. The author thinks that this distribution is possibly characteristic of zinc neuritis.

III. *Cases Dependent upon Fatigue of Certain Muscle Groups.* These are cases of occupation palsies in the narrower sense to which Edinger's exhaustion theory has been applied. In this group are also included cases in which, besides the fatigue, contributing causes are at work, such as alcoholism, diabetes, arteriosclerosis and tuberculosis.

Case 5. A railway employe received a blow on the right elbow which resulted in partial ulnar paralysis. For some years afterwards he worked as a ticket collector and constantly used his right hand, particularly the ball of the thumb, in punching the tickets. The right thenar eminence became flattened, and opposition and flexion of the thumb became diminished. The opponens and flexor brevis pollicis gave slow contractions both to electric and mechanical irritation. In other words, an occupational fatigue neuritis of the median nerve was engrafted on the traumatic ulnar neuritis.

Case 6 was a weaver with paralysis of the right musculospiral nerve and diminished sensation in the distribution of the second to the tenth dorsal nerves. In his work the extensors of the wrist and hand were overworked and the anesthesia on the thorax is ascribed to the constant rubbing of the clothing against the body, produced by the moving of the arm.

Case 7 was a female clerk, 20 years old, constantly engaged in writing, who developed pain in the right thumb, and later in the entire arm. She also developed tendon synovitis. Sensation was diminished in the lower third of the forearm and in the ball of the thumb.

Case 8. A male clerk constantly engaged in writing, gradually developed weakness and pain in the right hand. He developed atrophy of the hypothenar eminence and of the interossei. Abduction of the fingers was lost and adduction of the little finger was difficult. Sensation was diminished in the little and the ring fingers and in the ulnar side of the hand. While formerly writer's cramp was generally considered a functional neurosis, it has now become recognized that in at least many cases a neuritis exists.

Case 9 was a stone-mason who for 22 years had been working with a hammer weighing 13 lbs. in his right hand. He developed pain in the hand and sensory disturbance in the fingers, beginning in the little finger and gradually extending to the others. The patient was a heavy drinker and the author believes that the alcohol predisposed him to affections of the peripheral nerves, but the onesidedness of the neuritis and its occurrence in the habitually overworked muscle-area showed it to be essentially an occupation neuritis.

IV. *Cases of Neuritis Primarily due to Non-Occupational Causes, such as Alcohol and Infectious Diseases,* but in which the occupation to some extent determines the localization.

Case 10. The patient was a heavy drinker whose work caused great fatigue of the hand muscles, especially the opponens pollicis and the small finger muscles. He developed marked atrophy and weakness of these muscles while the other muscles, both in upper and lower extremities, remained strong. There was also pain in the legs and occasionally in the forearms. The reflexes in the legs became lost and the nerves in most places tender to pressure. There was diminished sensation in the hands, and in the legs below the knees. In other words, this patient suffered from alcoholic multiple neuritis in which the occupation caused an unusually severe involvement of the hands, while the lower extremities were comparatively mildly affected.

MULTIPLE NEURITIS.

Does Syphilitic Multiple Neuritis Exist? This ques-is answered in the affirmative by R. Demanche and P. J. Menard,[6] who define the disease as a multiple neuritis occurring in persons undoubtedly syphilitic who show no signs of mercurial poisoning, who have not been exposed to any other infection or intoxication capable of producing it, and in whom mercurial treatment does not aggravate the condition but rather tends to improve it. They report a case of a man, 46 years old, not addicted to alcohol, who had a chancre in November, 1908, and who, in July, 1909, developed bilateral facial paralysis of the peripheral type, accompanied by intense asthenia and Korsakoff's psychosis. A few days later the facial paralysis disappeared, but the patient developed flaccid paralysis, with loss of reflexes first in the lower and then in the upper extremities. Death ensued, preceded by a rapid rise of temperature and respiratory difficulty.

J. Hoffmann[7] calls attention to the fact that while the syphilitic affections of the central nervous system are exceedingly frequent and well understood our knowledge of the affections of the peripheral nerves is relatively scant. In the secondary stage we not uncommonly meet with unilateral or bilateral facial paralysis which is clinically indistinguishable from the ordinary rheumatic or refrigeration facial paralysis; and, occasionally localized neuritis of the ulnar, peroneal, or anterior crural nerves.

Syphilitic multiple neuritis has only reluctantly been recognized because the neuritic symptoms have been attributed to the toxic effect of mercury. In 1909 Steinert[8] could collect only 13 certain cases in the literature, and 15 other cases in which the neuritis occurred during or after the mercurial treatment, but

---

(6) Presse Méd., 1911, p. 930.
(7) Neurolog. Centralbl., Sept. 1, 1912.
(8) Münch. med. Wochenschr., 1909, p. 1938.

most of which undoubtedly were syphilitic rather than mercurial. Syphilitic multiple neuritis occurs without exception in the secondary stage of syphilis, from three to eighteen months after the initial lesion, at the time of the eruptions, glandular enlargements and specific sore-throat. It resembles other forms of multiple neuritis in its localization, tendency to symmetry and in the motor and sensory disturbances, but it has the peculiar features of being more prone to commence in the upper extremities, to lead to ataxia and to sphincteric disturbances. The pupils remain normal. So far no satisfactory anatomic investigations have been made.

Hoffman gives a detailed clinical report of a case in a man 21 years old who denied syphilis. In February, 1909, he developed a numb feeling in the hands and some weeks later in the feet. In April he began to have difficulty in using the hands, and in May he could not walk without a cane on account of unsteadiness, but there was no feeling of fatigue. There was no pain. When examined on June 1 he showed marked ataxia of the arms, legs and trunk, together with tremor of the arms. All tendon reflexes were absent. Joint and muscle sense, as well as ordinary forms of sensation, were diminished in the forearms and hands and in the lower extremities from the lower third of the thigh down to the toes. There was no marked loss of muscular strength and the muscles were not tender, but there was tenderness of the brachial plexus and the sciatic nerves. The electric reactions of the muscles were normal.

In July he developed some weakness in the leg muscles and loss of control of the rectal sphincter. The anesthesia of the lower extremities extended up to the groins and that of the upper extremities to the axillæ. In August there was paresis of all four extremities and swallowing became difficult. The nerve-trunks of the arms became thicker and harder, with slight spindle-shaped enlargements. He also developed bilateral facial paresis and tenderness of the nerve-trunks of the arms and legs. In September there was paralysis of the extensors of both wrists and hands. In

November a Wassermann test was made which was strongly positive. The cerebrospinal fluid showed an increase in albumin and marked lymphocytosis.

Mercurial treatment was commenced and the patient soon began to improve. When discharged in May, 1910, he still had a slight atrophic paralysis of the hands, paralysis of the peroneus muscles, slightly diminished sensation at the end of the extremities, loss of ankle-jerks and steppage gait. The tenderness of the nerve-trunks had disappeared. In October, 1910, the Wassermann test on the blood was negative and the patient could write on the typewriter, but his handwriting was tremulous. There was still paralysis of the peroneal muscles. An intramuscular injection of salvarsan was made. In August, 1911, he could write as well as ever and walked without a cane, but still had a partial foot-drop. The author feels certain of the diagnosis, largely on account of the palpable thickening of the nerve-trunks, which indicates an interstitial inflammation, so the affection of the peripheral nerves could not have been mere secondary degeneration from involvement of the roots in connection with syphilitic meningitis.

[The author does not hesitate to consider this a case of peripheral neuritis although the spinal fluid showed marked lymphocytosis and points out that there may be increase in albumin in the spinal fluid in diphtheritic neuritis. In the absence of anatomic examination, however, it is impossible to deny the existence of syphilitic meningitis. But that there was also a true multiple neuritis must be considered proven.—Ed.]

Beri-Beri. The following is the author's abstract of part of a paper read before the Scandinavian Medical Society of Chicago in September, 1912, by Dr. Axel Holst, Professor of Hygiene in the University of Norway. He has carried on extensive investigations of this disease and of scurvy which are of great importance to a sea-faring nation, like the Norwegians.

"The disease called beri-beri first of all occurs among the natives of tropical countries, such as Brazil, India and Japan. In addition to cardiac weakness, which is the most common cause of death, it presents as its most characteristic feature symptoms of polyneuritis.

This appears particularly in the lower extremities, and causes anesthesia, formication, numbness and paralysis. In the so-called 'dry beri-beri' the neuritis is very pronounced and accompanied by extensive paralysis and marked muscular atrophy, while there is only little edema. On the other hand, in the so-called 'wet beri-beri' the neuritis is in the background and is discovered only on careful examination for palsies and paresthesias. However, it is rare not to find some paresthesia, anesthesia and loss of knee-jerks, as well as some degree of foot-drop. There is marked edema, especially in the lower extremities. The cardiac weakness is about the same in both forms.

"It had for a long time been assumed that this disease had some relation to ingestion of rice, and about twenty-three years ago the Dutch physician, Eijkman, in Java, observed that chickens fed exclusively on polished rice died with symptoms of polyneuritis. Later this was found to be true of pigeons and ducks. But the disease does not set in when the outer layers of the rice are added. Later extensive investigations, also on human beings, have been made in French Indo-China and the Philippines, which show that when inmates of prisons or asylums or gangs of laborers for months are fed chiefly on rice, many cases of beri-beri appear, but if the polishings (outer portions) are added, the disease is avoided. For this reason so-called cured or parboiled rice has been used with great success to prevent and cure the disease. Other foods, particularly a certain bean, *Phaseolus radiatus,* also prevent and cure beri-beri both in fowls and man. It has been found that in the outer layers of rice and in the beans mentioned there are certain substances of unknown chemical composition, but which may be extracted by dilute acids and which are essential for the nutrition of nerves.

"The beri-beri occurring in Norwegian sailors, which has been studied by me, generally appears as 'wet beri-beri,' with slightly marked paralysis and marked edema, while that observed in residents of the tropics and Japan is generally the dry form. The disease usually appears on long voyages, for instance, from the tropics to Europe, or from the Gulf of Mexico to Argentina. It is most frequent on sailing vessels when the victims have been deprived of fresh provisions for a long time. Crews of whalers in the Antartic ocean, which for eight or nine months are dependent on provisions from home, are frequently attacked.

"This disease seems to be more frequent on Norwegian ships than on those of other nations. Thus, in 1900, of 200 English sailing-vessels which touched Falmouth, in England, only one was infected, while four each of 60 German and 97 Norwegian ships harbored the disease.

"The disease was practically unknown in Norwegian ships before 1894, when a new law governing provisions took effect. Rice is scarcely used on Norwegian ships, and cannot, therefore, be responsible. The chief article of diet is bread, and before 1894 the bread was made of rye, and pigeons fed with such bread do not develop the disease. Since 1894 bread made of wheat flour or a mixture of one-third rye and two-thirds wheat is used. This bread was found invariably to cause the disease in pigeons, and more rapidly with the bread baked of wheat flour alone. If the outer layers of wheat are added, the disease is prevented, as is the case with rice. It may be said that French and American sailors also use bread of fine wheat flour and yet do not develop beri-beri, but it is found that these people use other foods which neutralize the harmful effects of the bread.

"Other changes established in 1894 are also of importance. Before 1894 the Norwegian sailors five or six times a week ate salt meat, boiled at 100° C., but after this year they began to use canned meat and canned fish-balls, which before canning had been boiled at 100° C. to 120° C. Experiments show that chickens fed exclusively on such meat develop polyneuritis, which is not the case when the meat has been subjected to a temperature of only 100°. Furthermore, before 1894 Norwegian sailors ate peas four to six times a week, but after that year the peas were reduced to once or twice a week. This is noteworthy, as feeding experiments show that ordinary ship peas, even when boiled, prevent polyneuritis in fowls as certainly as the beans used in Java. As these beans also prevent and cure tropical beri-beri, the Norwegian government has sent large quantities of peas to Java in order to have them tried on beri-beri patients. So far only a few experiments have been made, but they have been very favorable."

After discussing the causes of scurvy and its relation to various forms of diet, Holst expresses himself as follows on the question of the relationship between scurvy and beri-beri:

"I will first mention that the same fresh vegetables, such as cabbage, which prevent scurvy in men and in the guinea-pig, also prevent and cure ship beri-beri, and as we have found in Christiania, they also prevent the experimental polyneuritis of fowls which represents the starting-point for our modern beri-beri investigations. Furthermore, pigeons or chickens fed on ordinary rice die of polyneuritis, *i. e.*, beri-beri; but guinea-pigs fed on rice die of pronounced scurvy and they show little or no neuritis. If pigs are fed on rice they develop both scurvy and polyneuritis and die.

"On the other hand there are facts which speak against the identity of the two diseases. It is true that like the extractives of rice made with dilute acids, the antiscorbutic substances of cabbage leaves may be similarly extracted, but there are certain striking differences of which the following will be mentioned. While ordinary ship peas are powerful in preventing the polyneuritis caused by rice in fowls, such peas do not prevent scurvy in guinea-pigs, nor scurvy in man. But if the peas are moistened and allowed to germinate, they are powerfully antiscorbutic in guinea-pigs as was shown by Dr. Fürst in Christiania who also found the same to be true of barley and oats. Guinea-pigs fed on these grains in the ungerminated state invariably die of scurvy, but when the barley or oats germinate they become powerfully antiscorbutic. This does not appear to be true of experimental polyneuritis. Thus the prophylactic effect of the previously mentioned bean, *Phaseolus radiatus,* according to the well-known beri-beri investigator, Grijus of Java, is lost when the beans are allowed to germinate.

"Under these conditions, we of the Hygienic Institute of Christiania must limit ourselves to the neutral expression that somewhere there is probably a bridge between scurvy and beri-beri, without stating where and what it is. It might, for instance, be assumed that fresh cabbage both prevents the experimental neuritis of fowls and the experimental scurvy of guinea-pigs because it contains two different substances. We have thought we might come closer to the solution of the question by observing if the osseous system of beri-beri patients presents the changes characteristic of scurvy. We have examined the ribs of 30 native beri-beri patients who died in Java and whose bones were sent to us through the kindness of a Dutch physician, and no such changes were found. However, only a few of these patients were in the twenties and most of them were considerably older. This is of some moment as the scorbutic marrow-changes occur at the age when ossification still is going on and therefore do not occur after the age of about eighteen years. But we did find in every case one bony change which is common in scurvy though not as characteristic as the marrow-change, namely a marked atrophy of the compact bony substance."

Experiments on the Causation of Beri-Beri performed at the Lister Institute of Preventive Medicine, are related by E. A. Cooper and Casimir Funk[9] who present their experiences as follows:

We have repeated feeding experiments of former workers upon birds (fowls and pigeons) with the various kinds of rice and have confirmed them, and have also confirmed the preventive action of rice bran (polishings). Experiments with an exclusive diet of various pure carbohydrates — starch, inulin, cane-sugar, and dextrin—have been carried out, and it was found that such diets induced polyneuritis with the usual symptoms. These results disprove any intoxication hypothesis concerning beri-beri in birds. A daily ration of 0.5 gm. phytin administered to pigeons fed on white

---

(9) Lancet, Nov. 4, 1911.

rice did not prevent polyneuritis, and attempts to cure polyneuritis by the addition of phytin, edestin, casein, and egg-yolk were unsuccessful. As the latter two substances are relatively rich in organically combined phosphorus the negative results are important. One gm. of dried pressed yeast per day prevented the development of polyneuritis in fowls, and we were also able to confirm the curative properties of pressed yeast. Yeast-juice obtained by squeezing the ground cells with *kieselguhr* in a press was found to exert a curative effect like the original yeast. *We have also found that pressed yeast hydrolyzed with 20 per cent. sulphuric acid for 24 hours still retained its curative properties.* As all organic phosphorus compounds are completely hydrolyzed under such conditions, and phosphoric acid has been shown to be without influence, it is very improbable that the polyneuritis is the result of a deficiency in phosphorus compounds.

After confirming the results of Fraser and Stanton concerning the beneficial effects of the alcoholic extract of rice polishings, we precipitated a water solution of the extract by means of phospho-tungstic acid, and found that the active substance is precipitated entirely by this reagent and that the filtrate is completely inactive.

The curative effect of the precipitate decomposed by baryta in the usual way, which is wholly devoid of phosphorus and also free from carbohydrates or protein groups, was tried upon ten pigeons; it was found to be extraordinarily active and cured the birds even when they appeared moribund, striking improvement occurring in from three to ten hours. In several instances birds which were completely paralyzed and supposed to be dying in the evening were found next morning with but some trace of lameness.

Polishings appear to contain only a very small amount of the active substance, and for further progress a very large quantity of polishings must be dealt with.

"In conclusion, it appears that the polyneuritis induced in birds by a diet of decorticated and polished rice is not due to the deficiency of phosphorus compounds, but to the absence of some substance not of protein nature, devoid of phosphorus, soluble in water and acidulated alcohol, dialyzable, and precipitated by phospho-tungstic acid, and the precise nature of which is being at present investigated by one of us (C. F.)."

"**Neuritis Multiplex Cutanea.**" According to H. Schlesinger [1] this is an affection of two or more cutaneous nerves without involvement of more deeply situated nerve-trunks or of the central nervous system. Of isolated non-traumatic cutaneous nerve affections the so-called meralgia paresthetica, or neuritis of the external cutaneous nerve of the thigh, has received much attention on account of its frequency. In rare cases other cutaneous nerves are also involved. In 1900 Schlesinger reported five such cases, in two of which meralgia paresthetica was combined with affection of one other cutaneous nerve, in one case the palmar branch of the median nerve, and in the other case two intercostal nerves. In the other three cases the chief affection was in the superficial branch of the radial nerve, combined with involvement of certain cutaneous nerves in the legs. Next to the external cutaneous nerve of the thigh the superficial branch of the radial nerve appears to be most frequently affected.

The etiology seems to be varied. Gout, syphilis, and exposure to cold and wet have been the principal factors. All of the cases observed by the author have been in the male sex.

The intensity of the symptoms varies considerably, ranging from mere paresthesias to excruciating pain. At first there is generally pronounced hyperesthasia of the nerve-areas affected, but later there may be partial or complete loss of sensation. Hence, when the painful area is anesthetic to touch we have the condition described as analgesia dolorosa. The point of exit of the cutaneous nerve is often very tender to touch, and if the nerve is in any way subjected to pressure or traction during muscular work the pain is increased. Vasomotor symptoms may occur. The prognosis is uncertain and the duration may be of weeks, months or years. Schlesinger reports two new cases:

Case 1 is a physician, 46 years old, in whom exposure

---

(1) Neurolog. Centralbl., Nov. 1, 1911.

to cold and wet appeared to be the only etiologic factor. The disease commenced with severe pain in the areas of both external cutaneous nerves of the thigh, of the anterior cutaneous nerves on the left side, and the fifth and sixth intercostal nerves on both sides. Marked hyperesthesia was later followed by diminished tactile, pain, and temperature sense. Under the use of warm baths and salicylates recovery took place in six weeks. At the height of the disease the patient had to stay in bed for one week.

Case 2. A physician, 31 years old, has suffered for five years from marked paresthesias in the right thumb and forefinger and recently also on the left side. At times there is severe pain. There is hyperesthesia and at certain periods hypesthesia. The pain is slight in the morning, gets more severe when the patient works, and is always absent during the night. No cause could be assigned in this case.

NEUROFIBROMATOSIS.

Familial Von Recklinghausen's Disease. "Generalized neurofibromatosis is always congenital, often hereditary and sometimes familial." This terse statement by Feindel has been amply corroborated. Recently J. D. Rolleston and N. S. Macnaughton[2] have tabulated 22 hereditary and familial cases published since 1900 and add a new one.

A girl 13 years old who came to the hospital on account of diphtheria was found to have a nevoid growth involving the right half of the upper lip, a small nodule at the margin of the hairy scalp, numerous punctiform pigment spots, so-called *café-au-lait* patches and blue spots. Some of these lesions were arranged in a zosteriform fashion. Her intelligence was above the average. A sister of 5 years also presented numerous *café-au-lait* patches, and a brother of 10 years presented a few pigment patches on and between the shoulders and a larger patch on the left buttock. The other brother of 14 years showed merely a slight yellowish tinge of the skin which was likewise present in the other 3 chil-

---

(2) Rev. Neurol. and Psych., January, 1912.

dren. Both paternal grandparents had many nevi and the father had a large number of molluscus tumors on the trunk and a few lesions on the face and limbs. The tumor on the older girl's lip was removed and found to be a plexiform neuroma attached by a pedicle to the infra-orbital nerve.

It is interesting that the lesions in the two girls increased in size and number during the time they were under observation in the hospital on account of diphtheria, and the father stated that his lesions became more numerous after an attack of pneumonia. Previous cases have been reported of neurofibromatosis appearing after typhoid fever.

A Case of Pyloric Stenosis Caused by Neurofibromatosis is reported by R. Leriche.[3] A man 45 years old had developed tuberculosis at the age of 18 years and in June, 1909, he became thin and cachectic and began to suffer from pain in the region of the stomach and from vomiting. Like his father he was the subject of generalized neurofibromatosis and one of his daughters had had numerous pigment spots. A laparotomy was performed six months later when one tumor was removed from the pyloric region and another from the duodeno-jejunal junction. The tumors consisted of fibrous tissue, the pyloric one containing sarcomatous elements. The patient died a few weeks later and at the necropsy a few intestinal fibromata were found which had been overlooked at the time of operation.

A Sudden Eruption of Neurofibromatosis is rare but 12 years ago Pierre Marie and Courclaire reported a fatal case in an alcoholic man, 52 years old, whose skin became covered with tumors in the course of two weeks. More recently J. Sabrués and J. Dubourg[4] report the case of a man who at the age of 23 years had a fibroma removed from the trunk, and at 28 years of age developed hundreds of cutaneous neurofibromata in the course of a few days. The eruption was accompanied by headache, fever, bronchitis, asthenia and leukocytosis.

Pierret and Sergeant[5] describe a female microceph-

---

(3) Deutsche Zeitschr. f. Chir., 1911, p. 314.
(4) Gaz. heb. des Sci. Méd. de Bordeaux, 1911, p. 591.
(5) L'Echo Méd. du Nord, 1911, p. 592.

alic idiot with numerous pigmentary nevi and a few vascular nevi distributed over the whole cutaneous surface. Some of the lesions were pedunculated and resembled molluscum pendulum. There were no subcutaneous tumors. One brother, still alive, also a microcephalic idiot, had extensive zones of yellowish pigmentation on the shoulders and molluscum on the back. Another brother, who died in an asylum for idiots some years previously, had had tumors of the nerve-trunks.

Signs of Tetany such as increase in the electric and mechanical excitability of the nerves, and the facialis and leg phenomenon, but without any spontaneous spasms, have been described in a case of multiple peripheral and central neurofibromatosis by H. Schlesinger.[6]

A Case of Neurofibromatosis Involving Ten Cranial Nerves is reported by J. M. Beattie and A. J. Hall.[7] The patient was a girl 17 years old who had noticed a lump on one arm one year, and on the other arm six months, prior to admittance to the hospital. Four years previously she had suddenly developed right hemiplegia, and vision had been failing for two years. When admitted she was blind and deaf, childish in manner, restless, twitched her limbs and made grimaces. Vertical nystagmus, bilateral choked disc, left facial palsy, and paresis of the left external rectus were the most striking neurologic findings. There were several subcutaneous tumors in the axillæ and on both sides of the neck. There was a pigmented macule on the right temple. The necropsy revealed innumerable neurofibromata on nearly all of the cerebrospinal nerves. All of the cranial nerves with the exception of the first and sixth pairs were affected. There were several growths within the spinal canal.

THE SYMPATHETIC NERVES.

Ganglioma Embryonale Sympathicum, a peculiar type of malignant tumor of the sympathetic nervous

---

(6) Mitteil. d. Gesell. f. inn. Med. u. Kinderheilk. in Wien., No. 7, 1911.

(7) Proc. Roy. Soc. Med., (Path. Sec.), 1912.

system, is thoroughly discussed by Ludwig Pick.[8] The chief points about this unusual tumor are the following:

1. Ganglioma embryonale sympathicum is a typical tumor of the sympathetic nervous system. It belongs in a class of ''unripe' neuromas and takes its origin in the formative cells of the sympathetic ganglia.
2. It occurs especially in children, even in the newborn, but is also occasionally seen in adults. It is nearly always malignant, grows into the vessels and forms metastases.
3. It is most frequent in the adrenals, unilateral or bilateral, often in connection with multiple tumors of the liver, or with tumors in the skull or lymph-glands. On account of the extensive embryonal distribution of the sympathetic formative cells it may be encountered anywhere. In this author's case it occurred in the uterus. The tumors in the liver need not be metastatic as there are frequently sympathetic cells on the under surface of this organ.

DISEASES OF THE MUSCLES.

A Case of Dermato-Myositis, the first one with necropsy, is reported by F. E. Batten.[9] This appears to be the first case on record in a child, with full pathologic examination. The patient was a girl 9½ years old, and the disease lasted 15 months, during the greater part of which she was under observation. The symptoms first came on shortly after removal of tonsils and adenoids, and consisted mainly of periodic attacks of acute swelling of the muscles and redness of the skin, with subsequent induration of the skin and muscles after the subsidence of the acute attacks. Careful and very thorough examination gave negative results as regards any affection of the joints, blood, viscera or nervous system, and also no sign of syphilis, tubercle, *Trichinella spiralis,* or any form of organism which could be cultivated on ordinary media. Post-mortem

---

(8) Berlin. klin. Wochenschr., Jan. 1, 1912.
(9) Proc. Roy. Soc. of Med., 1912. Abstract in Rev. Neurol. and Psych., May, 1912.

examination showed the disease to be limited to the skin, subcutaneous tissues and muscles.

The most striking point about the muscles was that in the superficial portions there was great atrophy of fibers, while the deeper portions were practically normal. Those parts of muscles separated from the subcutaneous tissues by a thick tendinous sheath escaped affection, while those fibers lying in close contact with the subcutaneous tissues and along the intermuscular septa, were liable to be affected. Perivascular infiltration of the vessels with small round-cells was a striking feature, also thickening of the vessel walls or even actual obliteration. There was also infiltration of the muscle fibers with lymphocytes and regular accumulations of these cells, or "lymphorrhages," in places.

The cause of the disease is unknown. A toxin of exogenous origin is suggested and the fact that the child lived in close proximity to horses suggests the possibility of infection having been conveyed from them.

Is Amyotonia Congenita a Sequel of Acute Anterior Poliomyelitis During Fetal Life? This interesting disease in which the first post-mortem examination was reported by Spiller and which has been fully discussed in recent volumes of this series (1910 vol. p. 179, and 1911 vol. 10 p. 185), is now denied an independent nosologic position by Marburg[1] who believes that it is merely a muscular weakness following an attack of poliomyelitis during intra-uterine life. The histologic findings are those of a disease which has run its course, and consist of atrophy of muscle with proliferation of interstitial tissue and loss of cells in the anterior horns of the cord, with proliferation of glia. Marburg believes that his theory fully accounts for the clinical and anatomic findings. [Further proof seems desirable and may be obtained by applying the serum-test for poliomyelitis. If Marburg is right, the blood-serum of such children if injected into a monkey together with active poliomyelitic virus should protect the animal from the disease, while control animals would become infected. —Ed.]

Marburg reports one clinical case and discusses fully

---

(1) Arbeit. aus d. Neurol. Institut, Wien. Vol. 19, p. 133.

the states of the various forms of muscular atrophy in childhood.

Case 1. A boy three months old, of normal size at birth, was noted at the age of six weeks to be unable to move his hands and feet. When examined the child appeared of normal mentality. Only fingers and toes were actively moved. The extremities were flaccid, except for flexor contractures at the elbows, and the neck was also flaccid. The knee, ankle and abdominal reflexes were absent. There were no findings in connection with the cranial nerves. The chief gross findings at the necropsy were: Muco-purulent bronchitis, with bronchopneumonic foci; chronic catarrhal colitis; left-sided purulent otitis; patent foramen ovale; incomplete obliteration of the ductus Botalli; uric acid infarcts. Histologic examination of the nervous system showed slight fibrous thickening of the spinal pia, uneven loss of fibers in the anterior roots, particularly the cervical roots, while the posterior roots were normal. The main changes were in the anterior horns of the cord, all of which showed more or less loss of ganglion cells, and many of the remaining cells gave evidence of degeneration. The cells of the bulb and of the cerebral cortex were normal. The glia cells were increased in the anterior horns. The peripheral nerves and muscles also showed old degeneration of their fibers. There was no typical cell-infiltration in the muscles.

Case 2. A girl of six years had weak, flaccid extremities from birth, and did not attempt to walk until she was five years old. The muscles were atrophic with simple quantitative diminution in electric excitability, not a reaction of degeneration. Some contractures developed. The tendon reflexes were lost. A small piece of calf muscle was excised and merely showed some hypertrophic muscle fibers and increase in interstitial fat. [Curiously enough, in his extensive literature study the author has overlooked the report of a probable case of fetal poliomyelitis by Batten which was discussed in the *Practical Medicine Series,* 1910, vol. x, p. 135.—ED.]

Metabolic Observations on Amyotonia Congenita are related by J. C. Gittings and Ralph Pemberton.[2]

---

(2) Amer. Jour. Med. Sciences, November, 1912.

Their patient was a boy, fifteen months old, who showed the characteristic muscular weakness, without signs of muscular atrophy or of rickets. The tendon reflexes were absent and the muscles did not react to either faradic or galvanic currents. At first his condition was very poor but he improved, and at the age of twenty-one months the metabolic studies were made. Six months later he died of pneumonia. The necropsy findings were characteristic of the disease, the principal changes being found in the muscles. The case and necropsy findings were reported by Griffith and Spiller.[3] (An abstract of their report and reproduction of some of their illustrations is to be found in the *Practical Medicine Series,* 1911, vol. x, p. 185.)

The metabolism was studied with respect to the urinary nitrogen, fecal nitrogen, urinary calcium, fecal calcium and the kreatinin. For control the metabolism of two normal children of the same age was also observed. All three children were placed on the same diet and were kept on Bradford frames to facilitate collections. On account of the difficulties attending such experiments definite results were obtained only in regard to the kreatinin, and fairly definite results in regard to the calcium. The average kreatinin output was greatly reduced, averaging 0.0166 gm., while in the control cases it was 0.0786 and 0.089 respectively. In the only other case of this disease subjected to such investigation, namely that of Spriggs, the kreatinin output was 0.0627 gm. The "kreatinin coefficient," which is the daily kreatinin elimination per kilogram of body weight, was as follows in the cases in question:

The authors' amyotonia case	0.66
Spriggs' amyotonia case	4.0
Normal child No. 1	7.04
Normal child No. 2	8.04

In the case of calcium, while the experiment was not entirely satisfactory, the result was sufficiently definite to indicate that there was no marked disturbance.

Myotonia Congenita, or Thomsen's Disease. Leonard Findlay[4] of Glasgow, reports a case of this dis-

---

(3) Amer. Jour. Med. Sciences, August, 1911.
(4) Quart. Jour. of Med., July, 1912.

ease in a man 28 years old, who had developed the characteristic muscular rigidity at the age of six years. There was no similar case in the family. The patient was a street-car conductor and had little trouble on account of his affliction, his only complaint being that it made him "look foolish" when he walked upstairs with other people, as he would lag behind. It was noticed that "he might make the first step all right but, on trying to flex the other leg to accomplish the next move, he finds that he cannot do so. He might spring off his foot to give himself the necessary impetus for the ascent, but his toes catch on the step, and he has to fall back to his old position until the spasm passes off, when he will mount the next step all right. But the same thing occurs with the other leg, and so on for the first two or three steps with each foot, after which the legs seem to become quite supple, and he runs up the remaining portion of the stairs as quickly as anybody. Sometimes he may have mounted a few steps before the spasm occurs, but as a rule it takes place during the first movements of ascent. If he had been walking for some time before, he might not experience this difficulty in bending his legs on climbing a stair."

The delayed relaxation of the muscles after contraction is well brought out by means of myographic tracings. Thus a series of successive tracings of the extensor muscles of the right forearm were made. The muscle was directly stimulated by striking it with a percussion hammer, and the contraction recorded by attaching to the middle finger a piece of thread and leading it over a pulley to a spring recording-lever. In this way the rise and fall of the hand were registered. By comparing these tracings with those of a normal person, it is seen that they do not become normal even after 31 successive stimulations. A similar behavior of the muscles was seen on galvanic stimulation (the so-called myotonic reaction).

In discussing the nature of the disease the author points out that in the only case in which necropsy was performed no changes were found in the nervous system. In the muscles, however, marked changes were found, both in this one case and in several others in which pieces of muscle were removed for examination during life. The fibers have been found to be unduly large, the transverse striation indistinct, the longitudinal fibrillation more marked than in normal muscle, and the sarcolemma nuclei increased in number.

However, the author does not believe that we are dealing with a primary muscular disease. He lays stress on the fact that the myotonic spasm appears more promptly in connection with voluntary movements than with involuntary or reflex ones. Thus, it has been shown that myotonia of the abdominal muscles invariably occurred on bending but not on defecation. Further, while tapping the quadriceps muscle or stimulating it with electricity would produce myotonia, reflexly produced contractions in eliciting the knee-jerks were normal. Studies of the kreatinin metabolism point to the muscles merely being hypertrophied, extra efficient, not degenerated. He therefore believes that the nervous system in some way plays an important rôle.

MISCELLANEOUS.

TOBACCO AND THE NERVOUS SYSTEM.

The Nervous Diseases of Tobacco Smokers is the subject of an exhaustive study by v. Frankl-Hochwart,[5] the distinguished neurologist of Vienna who has made notes of the amount of tobacco used by his patients for 16 years, and now possesses some 1,500 clinical histories of heavy smokers. He does not include cases in which alcoholism, diabetes, syphilis or other toxic conditions coexisted. In compiling his smokers he divides them into four classes and for purposes of comparison he considers three cigarettes to equal one cigar. The four classes are thus fixed:

1. Light. Up to 4 cigars or 10 cigarettes.
2. Moderate. Up to 7 cigars or 20 cigarettes.
3. Heavy. Up to 12 cigars or 30 cigarettes.
4. Excessive. More than 12 cigars.

Of 800 persons utilized in these statistics, 230 were non-smokers, 245 cigar smokers and 325 cigarette smokers; 22 per cent. were light smokers, 29 per cent.

---

(5) Deutsche med. Wochenschr., Dec. 7, 1911.

moderate, 9.5 per cent. heavy smokers, 9 per cent. excessive, while 30.5 per cent. were non-smokers.

About one-third of the smokers complained of symptoms which they themselves, though sometimes erroneously, ascribed to tobacco. Of the heavy smokers more than half had unpleasant symptoms of prolonged duration, chief among which were palpitation and general nervousness. Next in frequency was vertigo. And still less frequent were insomnia, tremor, fluttering of the eyes, dyspepsia, constipation and diarrhea. Some of these patients stopped smoking and described the symptoms then experienced which were attributable to the abstinence. Among these were pressure on the head, a feeling of intolerable emptiness of the head, and vertigo. Many had severe palpitation which was not rarely followed by a sensation of fear. The condition of the bowels was interesting. Many of the smokers had a bowel movement only after smoking a cigarette early in the morning, and in such people abstinence frequently resulted in constipation.

As to symptomatology, the cases are roughly divided in 4 classes or types:

1. *Cerebral type.* Headache existed in more than 20 per cent. of the cases, usually in the form of a dull pressure pain, but occasionally genuine migraine existed. The author found migraine appearing relatively late in life in 8 persons who had no family tendency to migraine, and who never had experienced headache before beginning to use tobacco. Noises in the head and vertigo were not uncommon in this class and the same is true of disturbance of sleep. One hundred and fifty patients complained of mental depression and fear. In some cases the latter was preceded by abdominal pain which the author suggests may possibly have been due to intermittent claudication of the mesenteric arteries. Fifty patients chiefly complained of impaired memory, but this in several cases really was nothing but the kind of distractibility which is so common in neurasthenics. Serious disturbances of memory are more likely to occur in children who use tobacco.

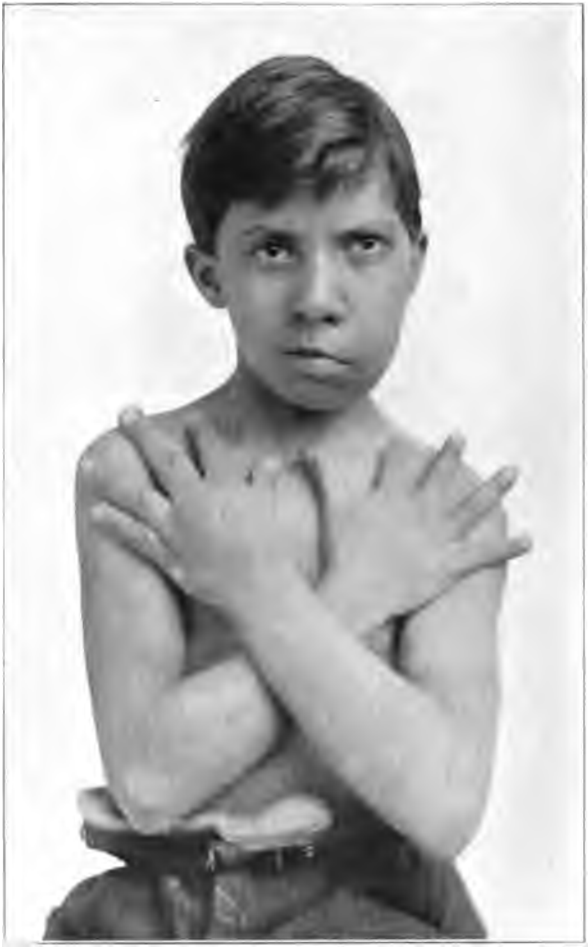
The author admits the existence of tobacco psychoses in rare instances such as the case of Pel. (Reviewed in *Practical Medicine Series,* 1911, vol. x, p. 230.) Transient losses of consciousness and syncopes occur, as well as attacks of apoplexy which may come on at an unusually early age. The author has seen 16 cases of the latter kind in which no other causes of arteriosclerosis existed.

While the rôle of tobacco in the etiology of epilepsy is not generally recognized among the Germans, Gowers and others admit it and Frankl-Hochwart had 14 epileptics among his heavy smokers. In 9 of these the disease existed from childhood and excessive smoking seemed to have had an unfavorable influence. In 5 cases the disease came on relatively late, between the ages of 24 and 40 and in the absence of hereditary tendency, organic disease or other toxic factors.

The symptom-complex of serous meningitis sometimes seems to be caused by tobacco, and the author speaks of this as nicotine meningitis. Two cases are cited. One was a lawyer, 57 years old, who was a heavy smoker, consuming as many as 14 strong cigars a day. He was suddenly taken with extremely severe right-sided headaches associated with nausea, vomiting and outbreaks of anger. No fever or other objective findings were present. The patient rapidly recovered and has remained well for two years afterwards. The second patient was a woman artist, 24 years old, who was admitted to the psychiatric clinic with signs of mental confusion. She had had an abortion a few weeks before. The onset was sudden, with severe headache and a sensation that the room constantly whirled around. Meningitis was first thought of but lumbar puncture was negative. She recovered in a few days. For a year before the onset she had smoked large quantities of cigarettes. Several months after her recovery she resumed smoking and again experienced a similar nervous attack following which she discontinued the habit.

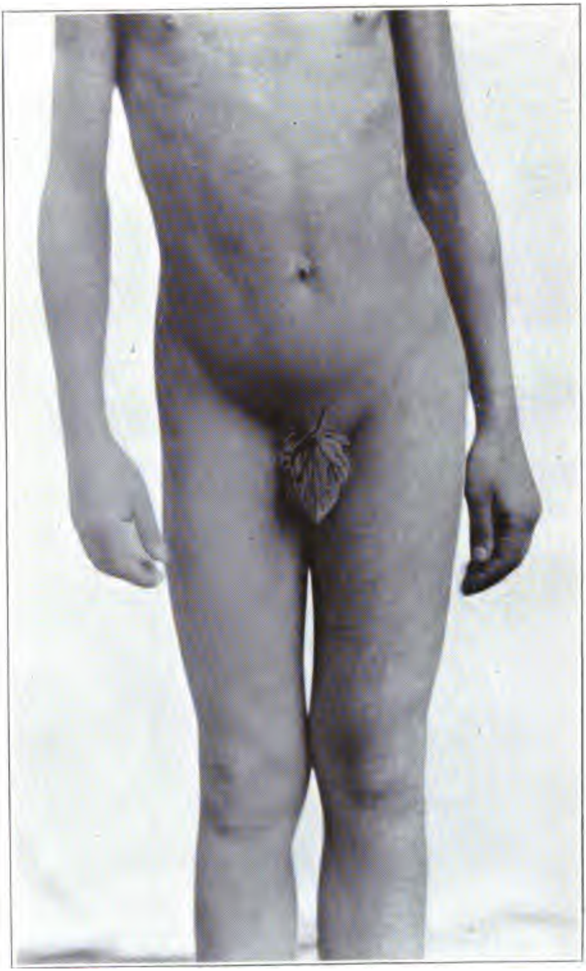
Of local brain lesions there were 11 cases which the author considers to be instances of intermittent claudication of the cerebral arteries, analogous to the ordinary intermittent claudication of the legs which Erb long ago recognized as being peculiarly frequent in smokers. Among the transient symptoms produced in

PLATE XV



Hypertrophy of left side of body.—Bassoe. (Page 189.)

PLATE XVI.



Hypertrophy of left side of body.—Bassoe. (Page 189.)

these attacks were aphasia, hemianopsia and hemiparesthesia. Aside from the well-known optic nerve affection causing tobacco amblyopia, the author found pupillary anomalies in 40 cases. In 22 cases there was some inequality of the pupils and in 8 cases sluggish reaction to light. Uhthoff believes that nicotine may cause a loss of the pupillary reflex but Frankl-Hochwart has not had any case of this kind in which other causes could be excluded. Cases of nervous partial deafness and even genuine Menière's syndrome may be caused by excessive smoking. The author has observed a unique case of intermittent deafness in a heavy smoker and he is inclined to attribute this condition to intermittent claudication of the auditory artery. He also has the impression that excessive use of tobacco may be one of the contributive causes in trifacial neuralgia.

2. *Spinal peripheral type.* Here neuralgic phenomena predominate. In 31 cases sciatica existed and in 23 cases there were painful affections of the nerves of the arms, of a kind resembling the so-called neurasthenic pseudoneuralgia described by Oppenheim. Diffuse pain is much more frequent than in the localized forms. Whether a tobacco multiple neuritis exists is a difficult question, but at any rate it appears that tobacco contributes to the appearance of neuritis when other factors, such as alcoholism, coexist. This has previously been pointed out by Oppenheim.

3. *Nervous affections of the internal organs.* The tobacco heart is well known, the chief symptom of which is tachycardia. Pseudo-angina, with marked states of fear is also common. Symptoms of nervous dyspepsia are common. Transitory glycosuria existed in 10 of the author's heavy smokers. The depressive effect of tobacco on the genital functions has long been recognized, though it is not mentioned in most textbooks. Changes in the testicles of dogs given tobacco in their food have been described. Seventy of the author's male patients complained of anomalies of genital function and it is also known that menstrual disturbances and abortions are common in women employed in cigar factories.

As to prophylaxis, the author does not condemn the moderate use of tobacco. He says: "The healthy person may safely go on smoking. I consider 4 or 5 light cigars or 10 or 12 light cigarettes perfectly permissible." No one should smoke regularly before the age of 17 or 18 years, and people with a tendency to heart disease or with signs of arteriosclerosis, diabetes or syphilis should be very moderate.

In the discussion of this paper which was read before the Society of German Neurologists, Erb reiterated his observations on the important rôle of tobacco in intermittent claudication.

The Toxicology of Nicotine was discussed at the same meeting by Frölich,[6] who states that nicotine is the only constituent of tobacco smoke to be considered, because the others are either non-toxic or, like carbon monoxid and prussic acid, exist in too infinitesimal quantities to be of any moment. Only a small amount of nicotine is absorbed. Acute fatal nicotine poisoning from smoking almost never occurs. In severe experimental nicotine poisoning excitement of the respiratory and cardiac centers is followed by paralysis. Nicotine is an energetic vasoconstrictor by its action both on the vasomotor center and on the peripheral vasoconstrictor ganglion cells.

General muscular cramps are also produced in experimental poisoning and glandular secretion is stimulated. Nicotine further acts upon striated muscle by producing a state of tonic contraction in the sarcoplasm. Long-continued use of tobacco leads to chronic poisoning and the tolerance produced is only a limited one. No tolerance to lethal doses is established. Habitual use of tobacco chiefly affects the vegetative nervous system. As there is only a slight degree of adaptibility to large consumption of nicotine, so the intensity of the symptoms produced by abstinence following excessive use is relatively mild and practically never assumes a serious character.

The frequency of arterial spasms in the chronic human tobacco poisoning is due to the strong effect of nicotine on the arteries. Degenerative changes in the optic nerves and retina are also experimentally estab-

---

(6) Deutsche Zeitschr. f. Nervenheilk., Vol. 48, p. 359.

lished results of chronic poisoning. The obtained results in attempts at the production of arteriosclerosis by injections of nicotine in animals should not be used in estimating the rôle of tobacco in human arteriosclerosis. The undeniable action of tobacco in producing abortion in tobacco workers is explained by its effect on all organs possessing smooth muscle innervated by the sympathetic nervous system. The high toxicity of nicotine should call for a propagandum for the introduction of smoking material from which nicotine has been removed.

HEMIHYPERTROPHIES AND HEMIATROPHIES.

Hemihypertrophy of the Body. This rare congenital anomaly has recently received considerable attention, particularly through the appearance of a thorough study by Stier[7] who also discusses the opposite condiof hemiatrophy, both that affecting the whole half of the body, and the more common facial hemiatrophy. He reports one case of hemihypertrophy of the entire right half of the body in a male child 4½ months old. No other case of malformation of any kind was known in the family. The birth was normal. It was at once noted that the right half of the baby's tongue was greatly enlarged, and a few weeks later the mother noticed that the right cheek was fuller than the left. At the age of three months the right leg was distinctly longer than the left and at four months the right arm was discovered to be the larger. Meanwhile, the proportion between the right and left halves of the tongue remained the same.

When examined at 4½ months, the baby was 63 cm. long. There were no abnormalities of the skin or of the eyes. The circumference of the chest was 1.5 cm. greater on the right side, and that of the abdomen 1 cm. greater. Both the right upper and lower extremities were 1 cm. longer than the left, and the circumference of the right thigh and calf, 2 cm. greater than the left. The circumference of the arm was ¾ cm. and that of the forearm 1.5 cm. greater on the right side. The ears showed no measurable difference. The mental development was normal.

---

(7) Deutsche Zeitschr. f. Nervenheilk., Vol. 44, p. 21.

Stier also reports a case of hemihypertrophy of the left side of the face in a boy 12 years old in whose family there had been no other case of malformation. The mother had died of tuberculosis. At birth the patient's left eye was enlarged and at the age of 6 months an iridectomy was performed. As the eye continued to grow larger, it was enucleated when the boy was 3 years old. Although there had been no difficulty at birth, the child's development was slow. He did not walk or talk until 3 years old. He went to school at 8 years but made poor progress. After the enucleation of the eye the eyelids and cheek on the left side grew disproportionately large and a precocious growth of hair appeared on the left cheek and upper lip. The bones of the left side of the face also became enlarged.

When examined at the age of 12 years the boy was of usual height, and the lower extremities were of equal size and normal in every respect, except for the presence of a few brownish pigmented areas in the skin of the thighs. Such areas were also seen on the trunk. The left ring finger was a trifle enlarged, otherwise the upper extremities were equal and normal. The boy was right-handed. The enlargement of the soft parts of the left side of the face was so marked that the nose and mouth were pushed over to the right. Aside from the great swelling of the eyelids in this case, the appearance of the face is very similar to that of Bassoe's case of hemihypertrophy to be related below, and a picture of which is found in this volume. The tactile and pain sense was diminished in the area supplied by the left trigeminal nerve and the left facial muscles showed a slight quantitative diminution of the electric reactions.

A piece of skin removed for histologic examination showed a picture identical with elephantiasis, namely a flattening and almost complete atrophy of the papillæ and infiltration of all cutaneous layers with small, round and spindle-shaped connective-tissue cells. The alveolar process of the left side of the upper jaw was greatly enlarged and more than twice as broad as the right. Nevertheless, the development of the teeth on the left side was backward, only one molar being present. The left ear was 6 cm. long, the right 5.5 cm., the region of the left tragus markedly thickened.

Stier reports a similar case of right-sided hypertrophy of the face in a boy 5 years old whose right eye was enlarged at birth and was enucleated at the age of 15 months. Before the operation the entire right side of the head and face had become enlarged, particularly marked enlargement being noticed in the zygoma and vicinity. The teeth were smaller on the affected side.

The entire literature relating to the subject is carefully reviewed and the various theories regarding the pathogenesis are discussed. Attention is called to the fact that the majority of cases of hemihypertrophy, both general and facial, have affected the right side while the majority of cases of facial hemiatrophy have been on the left side. This fact and theoretic considerations lead Stier to believe that the cause of the affection must be sought in the subcortical or cortical centers. Designating the left cerebral hemisphere as "superior" in right-handed persons and the right one as "inferior" in right-handed, but "superior" in left-handed persons, he formulates the following dictum: The excess formations and partial hypertrophies have their seat in the side of the body innervated by the "superior" hemisphere, while the defect malformations and hemiatrophies occur in the lateral half innervated by the inferior hemisphere. In this connection Stier calls attention to interesting statistics concerning various local defect and excess malformations in relation to the "superior" hemisphere. Thus, supernumerary nipples were found 27 times on the right side and 5 times on the left side in right-handed persons, and the only 5 cases in left-handed subjects were on the left side.

A Case of Hemihypertrophy, involving the entire left side of the body, is reported by Peter Bassoe.[8] The patient is an Italian boy whose left side has been enlarged since birth. Every measurement shows an enlargement on the left side. The nostrils, teeth, gums, ears, etc., are larger (Plate XV). There is a marked nevoid condition of the skin of the hypertrophic side.

---

(8) Amer. Jour. Insan., July, 1912.

The patient's mentality and general health are good. The thyroid is hypoplastic.

A fair number of very similar cases have been reported, so it is possible to make some generalizations. Heredity plays no rôle. The male sex and the right side seem to predominate. The hypertrophic side is generally the stronger. Nevi and pigmentation of the skin are very frequently present. The hypertrophy mainly involves the subcutaneous tissues, muscles and bones, namely, tissues of mesoblastic origin. In the few cases in which post-mortem examination has been made, no brain or other lesion has been found which throws any light on the cause of the condition. In a few of the cases enlargement of a few fingers and toes on the otherwise normal side was noted, so the enlargement of the entire right foot in the present case is not an absolutely unique feature.

A recently reported case, with necropsy, is that of Cagiati.[9] A female child which died of enteritis at 11 months showed enlargement of the entire right side from birth. There were nevi of the skin. The brain showed no gross change and the thyroid was normal. The left ventricle was hypertrophied, and the carotid, femoral, radial and other arteries were wider and thicker on the left side. The left lung was larger than the right, and all the bones on the left side were relatively enlarged.

Facial Hemiatrophy is also most lucidly discussed by Stier.[1] He reports a typical case in a girl 11 years old whose birth had been easy. She walked at 1½ years and stammered at first. She was decidedly left-handed. At the age of 9 years she became nervous, slept poorly and half a year later atrophy of the right half of the face set in. The skin and bones on the right side became thinner and the right palpebral fissure slightly wider than the left. By tabulating the reported cases Stier finds that 94 cases affected the left side and 47 the right side of the face. This agrees well with his theory that the atrophies and other defect malformations affect the side innervated by the "inferior" hem-

---

(9) Deutsche Zeitschr. f. Nervenheilk., 1907, Vol. 32, p. 282.
(1) Deutsche Zeitschr. f. Nervenheilk., Vol. 44, p. 21.

isphere; that is, they occur most frequently on the left side in right-handed persons and *vice versa* (as in the case just cited).

Statistics also show that the affection generally begins in childhood, most frequently between the ages of 10 and 12 years, and 75 per cent. of 109 cases set in before the age of 20. The disease commences at the same period of life as Friedreich's ataxia, muscular dystrophies, and other hereditary degenerations, and Stier is inclined to place facial hemiatrophy among the diseases dependent on "circumscribed hypofunction of inherently inferior tissue elements." Formerly the affection has been ascribed to disturbances of the sympathetic (Jendrassik), local infections (Moebius), lesions of the trigeminus and Gasserian ganglion (Mendel), affections of the peri-ependymal gray matter of the pons and bulb, "syringo-encephaly" (Brissaud). There are serious objections to all of these theories, such as the lack of proof of the existence of trophic fibers in the trigeminus and the occasional involvement of structures outside of the trigeminal area.

A Case of Hemiatrophy from Scleroderma. P. C. Knapp[2] states that congenital hemiatrophy as a rule is due to some malformation or defective development of one-half of the brain, occurring in fetal life. The much more common acquired hemiatrophy is generally the result of some cerebral lesion occurring in childhood and is associated with hemiplegia. He finds only 6 reported cases of complete hemiatrophy due to scleroderma. His own case was that of a boy 18 years old, first seen in 1907. Two years previously he had had some sharp paroxysmal pains in the left leg followed by spasmodic clonic contractions of the muscles of the thigh, and slowly progressive wasting of the whole leg. At about the same time pigmented nodules appeared in the skin of the left side of the abdomen and of the left thigh and leg. The left leg grew weaker and he had "drawing" sensations and spasms in the abdominal muscle and in the left arm. Measurements showed that practically every part of the left side was smaller than the right, including the circumference of the head

---

(2) **Boston Med. and Surg. Jour., May 11, 1911.**

and chest. The greatest difference was in the lower extremities.

A Case of Hemiatrophy of the Body is reported by Geist.[3] A boy, the second illegitimate child of a healthy mother, was born in June, 1899. The father was a healthy soldier whose ancestry is unknown. Pregnancy and delivery were normal. At birth the entire left half of the body was noticed to be smaller than the right. The child developed slowly, and did not walk until over 2 years old. At the age of 7 years he was brought to a school for the feeble-minded as he could not even learn to count to 5 in the public school. At this time (January, 1907) he was 100 cm. tall (3 ft., 3½ in.). The circumference of the head was 46.5 cm. In March, 1910, he was 110.5 cm. tall (3 ft., 7½ in.). The circumference of the head was 47 cm.; right half 24.5, left 22.5; the left half of the face, the left orbit and the left half of the tongue were distinctly smaller than the right. The gums and mouth showed a similar disproportion. The skin was nowhere abnormally thin or thick. The extremities were also slightly smaller on the left side. Both testicles were descended. The left knee-jerk was readily obtained while the right one was very weak. The boy could not talk in connected sentences but could tell his name and age. He did not know the names of the days of the week in correct order but knew left and right. There was no paralysis or disturbance of sensation. He was decidedly left-handed.

The author decides that the left side is the abnormal one and the case consequently is one of unilateral dwarfism because the boy is undersized, the average height of a normal boy of 10 years being 127.3 cm. (4 ft., 2 in.). As the right side more nearly approximates normal and as there are no other pathologic features than the asymmetry, the author decides in favor of hemiatrophy of the left side rather than hemihypertrophy of the right side. There was a deformity of the left external ear but no abnormality of the skin is described. [In nearly all cases of hemihypertrophy there has been a nevoid condition of the skin on the hypertrophic

---

(3) Neurol. Centralbl., 1911, p. 122.

side.—Ed.]. It is very interesting that the patient's half brother, who is 2 years older, and of normal mentality shows a moderate hypoplasia of the left side of the face, while the rest of the body is symmetrical. The author suspects some connection between the malformation and the mental weakness as there was no birth injury or other postnatal etiologic factor.

ANGIONEUROTIC EDEMA.

A Fatal Case of Angioneurotic Edema with Necropsy is reported by F. Harbitz.[4] The patient was a sailor, 20 years old, who was found dead in his bunk when a physician was called to attend him. The entire head and the neck were extremely edematous. The lips and tongue were swollen and the mouth full of frothy fluid. The soft palate was so extremely edematous that the entrance to the pharynx was closed. It was elicited that the patient previously had had several attacks of swelling of the head and neck and difficulty in swallowing. At one time there had been swelling of the right wrist, but at the time of death there was no edema of the extremities. The last attack began the day before death with swelling about the left eye. A few minutes before death he sat up looking at his face in the mirror, then fell backward, asked for help in a hoarse voice, threw his arms about, frothed at the mouth and died.

On examination of the organs the mucous membrane of the larynx and trachea was found to be edematous and pale. There was no exudate, ulceration or hemorrhage anywhere. The mucous membrane of the pharynx was also extremely edematous, otherwise the organs were normal.

The family history is of great interest on account of the occurrence of several similar cases. A brother, 27 years old, had attacks commencing with edema of the face or extremities followed by pain and vomiting. The first attack occurred at the age of 7 or 8 years. For a time he had 2 or 3 attacks a year; later more frequently, especially in autumn and winter. Aside

---

(4) **Norsk Magazin f. Laegevid, 1912, No. 2.**

from the attacks, his health has been good. He went through his military service without any attack. He is married and has 2 healthy children. Another brother died at the age of 15 years of tuberculous peritonitis and 3 died in infancy. A grown-up sister is well. The father had similar attacks and died suddenly, heart failure being given as the cause of death. His wife had noticed that he was hoarse a few hours before death. The paternal grandfather and his two sisters also were affected.

Similar cases affecting many members of the same family have been reported. Thus Osler in 1888 described a family with 22 cases in 5 generations, with 2 deaths from edema of the larynx, and Ensor in 1904 reported a family with 49 cases in 7 generations with 12 deaths of edema of the larynx. In most instances, the disease was transmitted through a healthy father or mother. The whole subject of the hereditary type of this disease is reviewed by Bulloch[5] who tabulates 170 cases with 36 deaths from edema of the larynx.

Harbitz also reports the case of a medical student, 22 years old, who for the last 7 years has been having attacks of sudden edema of the eyelids, lips, neck and at one time both thighs. The duration varied from a few hours to one day. A sister of 18 years and the mother are similarly affected. The author refers to Schlesinger's term "hydrops hypostrophos" which includes all types of periodic transient edemas, such as Quincke's circumscribed edema of the skin, acute recurrent edema of the eyelids, many forms of nervous rhinorrhea, intermittent swelling of tendon sheaths on a nervous basis, many cases of pseudo-croup, bronchial asthma, intermittent vomiting, nervous polyuria, nervous diarrhea and hydrops articulorum intermittens. Harbitz believes that we should also include the so-called anaphylactic shock occurring after injection of a small quantity of a foreign serum ("serum-disease"). In general it must be assumed that the so-called idiosyncracies are closely related to the angioneuroses. They are frequent in nervous individuals and in families with neuropathic tendency, and often become mani-

---

(5) Eugenic Laboratory Memoirs, IX; The Treasury of Human Inheritance. Part 3, 1909, Angioneurotic Edema, p. 38.

fest during pregnancy or menstruation or after psychic or physical traumas. The idiosyncracy may be toward an article of diet or some medicinal substance. The symptoms resemble those of the ordinary angioneuroses, such as sneezing, itching, urticaria, conjunctivitis, hoarseness and asthmatic attacks.

Paralysis Agitans.

A New Diagnostic Sign, the Cog-Wheel Resistance of the Extremities, is described by Harold N. Moyer.[6] "The term 'cog-wheel' is proposed as in a certain sense descriptive of a jerky or intermittent resistance of the extremities, when the sign is looked for. It is elicited by the examiner grasping the wrist with one hand and steadying the arm with the other, above the elbow. Rapid flexion and extension of the arm is made. Instead of an even movement, without resistance, when there is no involvement of motility, one, two or, perhaps, three slight hindrances to the movement are experienced by the examiner, which communicates to the hands of the examiner a jerky feeling. This is not sufficient to stop the movement of the extremity, so that it may be seen, but it is readily appreciated by the hand of the examiner. After the extremity has been passively moved for a short time, the jerks will slowly disappear, to be followed by a return after a period of rest.

"The symptom is most easily elicited and is most commonly present in the upper extremity, though it is occasionally noted in the lower. When testing the leg, the same method is pursued as when examining the arm, the patient being conveniently seated on a table and the leg rapidly flexed and extended at the knee.

"It is obvious that this symptom belongs to the early period of rigidity in Parkinson's disease, and it is this fact that gives it its value, from a diagnostic point of view. The term "paralysis agitans" is a misnomer. No patients with this disease have typical paralysis and many of them are without tremor. The es-

---

(6) Jour. Amer. Med. Assoc., Dec. 30, 1911.

sential symptom in the disease is the rigidity, and this is frequently an early symptom, preceding the tremor, although in some cases there may be tremor without rigidity, or at least without sufficient rigidity to admit of its being recognized in a clinical examination. So far as I have observed, this symptom is not noted in other conditions; hence when obtained it is strikingly characteristic of Parkinson's disease.

"Its value, from a diagnostic standpoint, is due to its presence in the cases in which there is real difficulty in making a diagnosis. The ordinary typical case of paralysis agitans, with the pill-rolling movement, the festination, stooping, and mask-like features, can be recognized at a glance, but there are less developed cases characterized by rigidity and absence of tremor, and frequently in which the disorder is limited to one extremity, in which the diagnosis is difficult. It is in this class of cases that I have learned to appreciate the value of this sign. In a number of cases in which the disorder began in one hand, characterized only by clumsiness and discomfort in the extremity, this sign was elicited and later, after some years, the more frank symptoms of paralysis agitans developed."

The Condition of the Larynx and of the Ankle Reflexes in Paralysis Agitans was studied in 80 cases, 56 of whom were males, by Graffner.[7] There were three types of laryngeal conditions observed:

1. Participation of the vocal chords or of the entire larynx in the tempo of the general tremor; 21 cases or 26.25 per cent.
2. Tremor of the vocal chords in a tempo different from that of the general tremor; 27 cases or 33.75 per cent.
3. Lack of genuine vocal chord tremor; 32 cases or 40 per cent.

The tremor accompanied all phases of activity of the vocal chords in cases of paralysis agitans *sine agitatione*. It was not always possible to detect a genuine tremor of the vocal chords. It was not uncommon to find slowness of muscular action. In 12 cases the adduction, and in 5 cases the abduction, were particu-

---

(7) Berlin, klin. Wochenschr., 1911, No. 38.

larly affected. Of 34 paralysis agitans patients only 5 showed a normal pharyngeal reflex, while it was absent in 20 cases and diminished in 9 cases.

Since Kurt Mendel has described anomalies of the ankle reflex in this disease the author has tested it in 23 cases, only 3 of which had normal reflexes. In 7 cases they were absent on both sides, in 4 cases weak on both sides, in 3 cases absent on one side, in 5 cases weak on one side, in one case increased on one side and absent on the other. As controls 28 comparatively healthy old men were examined and 18 gave normal reflexes, which leads the author to conclude that the changes in the ankle reflexes are more closely connected with paralysis agitans than with mere arteriosclerosis.

In connection with the disturbances in the larynx the author calls attention to the work of Fritz Heinrich Lewy who has described a form of degeneration characterized by globular or elongated deposits which stain a deep red with Mann's stain, occurring in the dorsal vagus nucleus, and which was present only in patients who clinically have shown tremor of the vocal chords. Such deposits were not found in other nuclei.

Stellwag's Sign in Paralysis Agitans. This sign, which consists in enlargement of the palpebral aperture and infrequent winking is almost constantly present in paralysis agitans according to F. Schultze[8] who considers it of value in differentiating this disease from multiple sclerosis and hysteria. It is at least as frequent in paralysis agitans as in exophthalmic goitre, and is explained by the increased tonus of the levator palpebræ superioris or of Müller's smooth muscle. In one case involuntary winking occurred only about every five minutes.

Resection of Posterior Roots for the Relief of Rigidity in Paralysis Agitans has been performed by Leriche,[9] of the Surgical Clinic of Lyons. The patient was 53 years old and had complained of stiffness and tremor for 8 years. He had become addicted to morphine and was in a deplorable condition, and he and

---

(8) Deutsche med. Wochenschr., 1911, No. 44.
(9) Neurolog. Centralbl., Sept. 1, 1912.

his relatives were anxious to have an operation performed which could promise a chance of relief. The fifth, sixth and eighth cervical posterior roots were cut by Guleke's extradural method. The result was that the tremor became less marked but there was no change in the rigidity although the patient found it easier to move his arms. He was well satisfied and was able to reduce his morphine by two-thirds. The author believes that better results would be obtained by cutting the lumbar roots for the relief of rigidity in the legs.

While feeling that our general conception of Parkinson's disease would discourage any thought of successful surgical intervention he took courage from the following statement by Van Gehuchten:[1] "We are completely ignorant whether resection of posterior roots might have a favorable influence on the Parkinsonian tremor but we believe that it would relieve to a great extent the hypertonicity of the muscles, and thus enable these unfortunate patients to regain in part a capacity for voluntary motion. Such an operation in cases of great rigidity would appeal to us as highly commendable."

Metadromal Progression is a characteristic symptom of paralysis agitans according to F. Tilney.[2] While the spontaneous gait of the patient is very slow and uncertain, he can walk quite well when another person walks a short distance ahead of him. There seems to be a lack of initiative in regard to the forward movements.

SUNSTROKE.

The Nervous Symptoms Following Sunstroke were studied by T. H. Weisenburg[3] during the hot summer of 1911. He states that although the literature of nervous symptoms following heat intoxication or exhaustion is not very large, yet a great variety of symptoms have been described. Besides the usual rise of temperature, headache and sometimes coma, there have been quite a number of cases of motor lesions which

---

(1) Bull. de l'Acad. roy. méd. de Belgique, Dec. 3, 1910.
(2) Neurographs I, 111, No. 3.
(3) Jour. Amer. Med. Assoc., June 29, 1912.

have been either hemiplegic or paraplegic and rarely acute ataxia and disturbance of speech. Curiously enough he has not found a single instance of such lesions producing sensory disturbances, although there is no reason why this should not occur.

Not much is known of the pathology chiefly because of the rarity of necropsy studies. As Oppenheim states, it has been thought that there occurs cerebral hyperemia; others assume thrombosis and multiple capillary hemorrhages in the medulla, anemia of the brain, edema of the pia, meningitis, cell-changes, dehydration of the tissues, auto-intoxication, etc. Considering the fact that the symptoms come on suddenly after a rise of temperature, it is not improbable that there is produced an auto-intoxication which in some cases causes multiple hemorrhages. He reports 2 unusual cases in detail.

Case 1. The patient was brought to the hospital unconscious with a temperature of 107° F., which dropped to 103° F. after an ice-bath; but fever persisted for three weeks. During the first week he was unconscious and during the following three weeks he was more or less stuporous. He was so ataxic that he could walk only on his hands and knees. At times he became violent but later his mentality seemed to be normal. He had no headache, nausea or tinnitus. The eyes were normal. He was weak in the extremities, the tendon reflexes were increased and ankle-clonus and the Babinski sign were present on both sides.

The most striking condition about the patient, however, was his extreme ataxia. He would lie perfectly still but on attempting a sitting posture his head and shoulders would show the most exquisite ataxia. In fact even when lying in bed and protruding his tongue the movement was ataxic. Any movement of the arms and legs would bring about an extreme inco-ordination which was not influenced by closing the eyes. The patient could smile and was not at all emotional. His condition when he was removed from the hospital was only slightly improved from that when he was brought in, but his ataxia remained the same.

Weisenburg comments on the case as follows:

"The symptoms were indicative of a lesion in the cerebellum and in the motor columns on both sides. It is apparent that the motor columns were probably involved in the medulla because there was implication of the upper and lower limbs and not of the face.

"As far as the other symptoms are concerned it is possible to explain all of them by a cerebellar lesion. First of all, the so-called maniacal outbreaks were not so much maniacal as they were the result of temper. The man was a Pole and could not make himself understood and it was only when he was exceedingly annoyed that this would occur.

"His speech was not at all aphasic. Examination of the pharynx and larynx did not show any palsies and it is apparent that his inability to talk and articulate was the result of his cerebellar inco-ordination. It is interesting to consider that in this patient, while the whole body was ataxic as was also the tongue, yet there was no ataxia whatsoever of the eyeballs, neither was there difficulty in eating and swallowing. This would argue for a separate cerebellar center for co-ordination of the eyeballs which is in accordance with the view set forth by Victor Horsley, who advanced the opinion that in the intrinsic nuclei of the cerebellum the movements of the eyes and head are represented, while in the so-called paracerebellar nuclei in which are included Deiter's nucleus, the vestibular nucleus and the nucleus magnocellularis substantiæ reticularis, are represented the movements of the trunk and limbs specially."

Case 2. A cook 41 years old was taken with jerkings and contractions of the muscles in various parts of the body, then suddenly the muscles would contract, forming large hard knots over his stomach, upper and lower limbs, face and neck. They were so painful that the patient would curl up and during them could not walk or straighten out his limbs. By different forms of manipulation he would be able to relax the muscles but the spasms would come on again. At times because of the muscular contraction of the abdominal muscles he could not retain anything on his stomach and would constantly vomit. Again he could not talk or eat anything because he could not open his mouth wide because of the contraction of the muscles of the face and neck. His right shoulder-joint was dislocated a number of times by the force of the muscular contraction. An attack would last from three to four days. They were not always, however, of the severity described above and occasionally he had only a muscular contraction in different parts of the body and some tremors. As in the other cases, following the extreme muscular spasm he would have fibrillary twitchings in the muscles.

The interesting part about his history is that these attacks always came on while he was working in a kitchen and was overheated and would never appear any other time. Physical examination was entirely negative.

The author states that the commonest manifestation of heat intoxication is the occurrence of muscular spasms, which has previously been commented on by Edsall[4] who reported 4 cases. In one there occurred tonic spasms of the forearms, legs and abdomen which were exceedingly painful, lasting from one-half minute to a minute and were brought about either by direct stimulus of the muscle, by movement whether voluntary or involuntary, or by electrical stimulation. These spasms were over in about twenty-four hours and nothing remained excepting a slight exhaustion and muscular soreness, although fibrillary twitchings were seen in the muscles for a number of days following and these were present also between the spasms. The reflexes and sensation were normal and there were no other symptoms present. Edsall, as a result of his metabolic studies, believed that the spasms were the result of an acute degenerative process in the muscles. He stated that these spasms were in direct relation to the severity of the exposure and the degree of musculature. Sometimes they are so severe as to cause death. They may occur from ordinary exposure to high atmospheric temperature in the summer and are quite common among stokers in the navy.

---

(4) Jour. Amer. Med. Assoc., Dec. 5, 1908.

ELECTROCUTION.

Brain Lesions Produced by Electricity are described by Spitzka and Radash[5] who examined the brains of 5 electrocuted criminals. In all of the cases there were certain peculiar areas, mainly in the medulla oblongata, cervical cord and midbrain. These areas, unlike anything ever seen before by the authors, are circular in outline, ranging in diameter from 25 to 300 μ. The most marked areas measure from 150 to 200 μ. They are seen to consist of two portions, a central rarified part and a peripheral condensed zone. The most characteristic areas contain a blood-vessel (capillary or precapillary), surrounded by a delicate small-meshed reticulum representing the central four-fifths of the area. The fibrils of the reticulum are, in the main, radially directed, and nuclei are sometimes observed along this course. The peripheral zone surrounding the central portion in a ring-like manner appears to be condensed, staining more deeply than the surrounding unaffected tissue, and is sometimes seen to be composed of circularly arranged strata, which are usually free from nuclei.

"One can only guess at the mode of formation of these lesions. It seems inconceivable that they could be due to sudden momentary dilatations of the blood-vessels themselves. The bead-like arrangement of the lesions along the blood-vessels, the condensation zone limiting each lesion, and the radially disposed fibers and torn tissue would seem to indicate a sudden liberation of the bubbles of gas due to the electrolytic properties of the current as it seeks the paths of the least resistance along the vessels. The maximum number of lesions are found in the most constricted parts of the brain-stem in the path of the current, and are most numerous along the longitudinal fiber-tracts and blood-vessels."

PELLAGRA.

Beverley R. Tucker[6] states that in 1910 it was estimated that there were 5,000 cases of pellagra in the

---

(5) Amer. Jour. Med. Sciences, September, 1912.
(6) Amer. Jour. Med. Sciences, March, 1912.

United States, while in 1911 the number is thought to be at least 10,000. The author has studied the disease very extensively both inside and outside of the institutions for the insane, and in the present article dwells particularly on the nervous and mental symptoms. As to the former he says:

"The nervous syndrome of the pellagrin may be said to consist of restlessness, insomnia, muscular weakness and vertigo as almost constant symptoms, with headache, tremors, cramps, changes in the knee-jerks and pupillary reflex, sway in station and slowness of speech occurring in about one-half of the cases, while areas of cutaneous anesthesia, loss of sexual power, tenderness of the spine, and diminution of smell and of finger touch are found in about one-fourth of the cases. Trophic disturbances, contraction of the extremities, ataxia and paralysis sometimes occur. Many mild or early cases only show restlessness, weakness, insomnia and vertigo, while all of the severe cases with which I am familiar show deep reflex, pupillary and special sense changes before death.

"A study of the *mental condition* of these patients showed emotional irritability manifested either by excessive weeping or outbursts of temper in 53 per cent. of the cases. Depression was present in 84 per cent. of the cases and in about half of these the patients were said to have ceased laughing since the onset of the attack. Memory was distinctly poorer than usual in 74 per cent. of the cases. Delusions and hallucinations were present in about 20 per cent. of the cases. Most of the delusions were those of persecution and were unsystematized, and the hallucinations were the usual ones of sight and sound. These symptoms are much more common among institutional cases. Apprehension, varying from anxiety about their condition to delusions of impending danger, was present in 100 per cent. of the cases. Homicide was not attempted in any of these cases, but suicide was committed by 1, attempted by 2, and threatened by 6.

"The mental syndrome of these non-institutional cases shows apprehension, depression, emotional irritability and failing memory as common symptoms, with delusions and hallucinations and suicidal tendencies not infrequent. The mental symptoms, except apprehension, may be absent in mild or chronic cases and severe cases may occasionally go on to death without developing delusions or hallucinations. All of the severe cases show depression, emotional changes or failure of memory. The severe mental symptoms somewhat resemble melancholia, but they are usually accompanied by neurologic signs not found in melancholia. The melancholia depression attitude is not often assumed, the agitation shown by wringing the hands is not seen and delusions of unworthiness are not present. Some of the mental states are said to resemble mania, but I have never seen the flight of ideas, the chaotic delusions, the changes of personality, the exaltation or the increased strength so common in acute mania.''

MENTAL DISEASES.

GENERAL CONSIDERATIONS.

The Value of Psychology in Psychiatry is discussed by Adolf Meyer[6] whose closing remarks are well worthy of attention.

"Summing up the situation, I wish to emphasize my view that psychology will become a much more real issue when it aims to guide students in the correct and critical recording of the plain facts of conduct and behavior and the mechanisms at work, and when it does more to encourage them to take the common-sense attitude about psychology; when it puts the emphasis on what shows objectively as well as subjectively, namely, the conditions, types and results of mental activity; when it trains the student to see, in the special psychologic methods, methods of accuracy and greater comparability; but above all, when the data can be formulated in terms of adaptations of a living functioning organism with the system of mental integrations—the conditions and structure of which are becoming increasingly better understood. The student should learn that psychology is the study of a certain type of reactions, of the conditions under which they occur, of what they do and how they can be modified and kept from miscarriage. How to use the reactions either as mere signs of broader disorders, or as dynamic factors, must be learned in the study of cases and by experimentation. In this connection I should urge that we avoid depriving the field of its freshness and concreteness in courses to undergraduates by teaching abstract systems of psychopathology merely illustrated by the parade cases of the literature, and leaving them with dyspepsia when confronted with our

---

(6) **Jour. Amer. Med. Assoc., Mar. 30, 1912.**

plain hospital cases and the less sensational disorders. It is equally uncalled for to describe psychopathology merely as 'applied psychology.' We do not call pathology 'applied physiology,' or autopsy work 'applied pathology.' For the establishment of some fundamental facts the simple autopsy is sufficient; for others it is the starting-point of deeper work, possibly demanding an experimental reconstruction of the facts. In psychiatry the so-called clinical observation and clinical conversation and routine examination with its record in questions and answers is the basis for the various ramifications, and the more we cultivate safety in these ordinary starting-points the safer our position. Psychopathology is not applied psychology. It is a field from which psychology may expect just as much broadening out as psychopathology can expect help and experience from the methods of so-called pure psychology. If, for instance, we can work with dynamic principles in psychopathology, all the better for psychology as a whole; but even on neutral ground with methods already current in psychology, valuable collaboration is possible and offering friendly companionship of interests.

"I repeat: Psychology is the only field of experience from which we can get the proper designation and understanding of many reactions which we must learn to describe, to differentiate, to understand for the conditions under which they arise, for the share played by the different integrative factors of the reaction as a whole, for a reduction to terms of an experiment of Nature and for the study of its modifiability. Even in its most dogmatic form psychology offers methods and precautions to our workers; and the more we shall learn to know where we can safely depend on qualitative tests of our reactions and where quantitative tests are possible and worth while and the more we shall use the dynamic aspects of the facts, the greater will be the appreciation of a demand for psychology on the part of medical students. Psychopathology is not merely applied psychology, but can be a legitimate and fruitful field of psychology at its best."

The Teaching of Psychology in Medical Schools.

Shepherd Ivory Franz[7] pleads for the teaching of psychology in medical schools, and points out how the profession has suffered for the lack of it.

"There can be no doubt that much of the success of the followers of Christian Science, of 'mind cures' and of other fads and fancies, has been due to their recognition of the psychologic factors in the mental life, even though this recognition has been reached by unscientific reasoning, and without special training. It has been largely because of the lack of recognition of the psychologic aspect of disease by the medical profession that non-medical healers have been denounced, but, as Prince rightly remarks, these should be looked on rather as an evidence of lack of medical insight. Those who have charge of medical education must realize that students of medicine should not be permitted to pass through their course of medical preparation without at least an elementary knowledge of mental processes. It is necessary that the graduate in medicine be prepared to appreciate the power of mind for health or illness; that he be given at least a view of mental conditions, signs and therapeutics; and that he be prepared to treat many individuals who now seek with success charlatans of all kinds."

The Content of a Course in Psychology for Medical Students is tentatively outlined by John B. Watson,[8] director of the Psychological Laboratory in Johns Hopkins University. "I suggest that the course should require three periods each week; two laboratory periods of two hours each and one lecture, the course to continue for one full year, probably as an elective in the third year. I think that this should represent the minimum of time which should be devoted to psychology in the medical school. If the laboratory work is to be touched on at all, then let us do the work with some degree of thoroughness. Unless this amount of time can be given over to the work I should favor simply a lecture course covering the more important topics in psychology. While I fully realize the fact that psychology and psychologists have more or less wasted an enormous amount of time on the topic of sensation, I feel,

---

(7) Jour. Amer. Med. Assoc., Mar. 30, 1912.
(8) Jour. Amer. Med. Assoc., Mar. 30, 1912.

nevertheless, that some experiments should be given in the topic of sensation. I suggest, therefore, that inasmuch as the whole ground cannot be covered, we centralize our efforts in sensation on two senses—vison and hearing. With a proper apparatus for the mixing of spectral colors the work in color-vision, including the tests on color-blindness, can be accomplished in three weeks' time. If we have to send the student to the color-wheel and have him go through the endless mill described by Titchener, he will get discouraged and waste the better part of three months and still come out with a very limited knowledge of the main features in the study of vision.

"The study of auditory sensation, in so far as its data would be valuable for the medical student, can likewise be completed in a short space of time. One needs for this work a good set of forks, the Edlemann's testing-forks, a set of differential forks—the siren, the Lehmann acoumeter and some of the more common pieces of apparatus which every psychologic laboratory now contains. By means of these simple instruments we could give the medical student a command of the method of testing limits of tone, tone islands, pitch differences, difference tones and the like. The lecture-work going along with the study of sensation should condense the work in the sense fields. It should confine itself to the data and should leave out of discussion the endless details of color theories, and theories of audition. The emphasis should be placed on the function and use of these two sense-organs and on the methods of studying them.

"I think that this short course in sensation would give the medical student some notion of the proper setting and control of a psychologic experiment.

"These experiments on the sense fields should, in my opinion, be followed by a thorough study with laboratory and clinical work of the Binet-Simon tests, this work to be supplemented on the experimental side by such studies as Thorndike is making on the 'curve of work,' initial spurts in work, spurts after interruption, end-spurts, etc.—in other words, by tests which show the part that fatigue, disturbances and emotional factors play in the normal on-going of mental work. Although the work on 'fatigue' in the usual sense is in a chaotic condition at present, I am not sure that we are justified in leaving it out of our course. I have the feeling that if psychologists will interest themselves more in the acquisition and control of motor processes rather than in the 'pattern' or 'content of consciousness' when entering on studies in fatigue, there will be brought about a rehabilitation of the ergographic methods of experimentation which will be to the advantage both of psychology and medicine.

"In my judgment this work on mental tests should be followed by experimental studies in the acquisition and retention of skilful acts; these studies should be made under several different sets of conditions. This type of experimentation, showing the different stages and methods of acquiring accommodations, the distribution of effort in learning, the short cuts in learning, etc., should be of tremendous interest to the psychiatrist."

After this Watson recommends a study of the normal process of association, memory, and retention, and this study should be made as objective as possible. Finally some time should be given to experiments on normal reaction time.

Morton Prince [9] also advocates a course in psychology for medical students and wishes to include the following topics:

1. A study of the subconscious (whether regarded as psychical or physiologic) in all its relations. This embraces the content of the subconscious as the storehouse of conserved experiences; the influence and mechanism of the subconscious in determining conscious and physiologic processes; its incubations; its automatic manifestations—motor and sensory; its syntheses, dissociations, etc.
2. The phenomena of hypnosis and allied conditions.
3. Suggestion and its phenomena.
4. Memory as a process (as distinguished from its content and phases) and the part played by this process in conscious and subconscious activities.

---

(9) Jour. Amer. Med. Assoc., Mar. 30, 1912.

5. Amnesia and its mechanism.
6. Fixed ideas, conscious and subconscious.
7. The dissociations and syntheses of "personality" and their mechanisms.
8. The emotions and feelings as forces which determine conscious and subconscious processes, dissociations, syntheses and activity, and which control the visceral functions of the body.
9. The emotional instincts as impulsive forces which determine mental activity and conduct.
10. The sentiments as complexes of ideas and emotions, and the part played by them in determining so-called personality and reactions to the environment.
11. Phenomena of repression, resistance, conflicts, inhibitions, etc.
12. The mechanisms of thought.
13. Complex formation.
14. The influence of complexes as conscious and subconscious settings or contexts in determining judgments, attitudes of mind, points of view, the meaning of perceptions, etc.
15. Associative processes and reactions, including word and galvanic reactions.
16. Habit processes.
17. Automatisms.
18. The mechanism of dreams.
19. The influence of the mind on the body.
20. Fatigue.

The Value of Field Work in the Study of Heredity in Mental Diseases is the topic of an address by H. A. Cotton,(1) clinical director of the New Jersey State Hospital at Trenton. Heretofore the methods used in gathering data on this subject have been woefully inadequate. Cotton says that as far as this country is concerned we must admit that the stimulation to the adoption of scientific methods in the study of heredity has come from one outside the medical profession, namely Dr. Davenport. When he began to co-operate with institutions for feeble-minded and epileptics a new era commenced, and the work was taken up by such leaders as Goddard of the Institution for Feeble-Minded at Vineland, N. J., and Weeks of the Epileptic Village at Stillman. These men employed trained field-workers to collect their data.

---

(1) Ill. Med. Jour., October, 1912.

In only one other country has such work been established and naturally this is Germany, but it is surprising that although Mendel, the formulator of the laws on heredity which bear his name, was a German, only one German psychiatrist has instituted any systematic study of heredity, namely Rudin of Munich. His work is all the more remarkable from the fact that he has done the field-work personally and at his own expense. He gives up his position as physician at the Psychiatric Clinic at Munich so many months each year and travels through Bavaria to collect his data. Rudin's[2] article is the most complete which has appeared on the subject, containing the most accurate family pedigrees ever gathered and also the best digest of the literature to be found anywhere.

The work at the Trenton State Hospital has been in operation for a year with the aid of two trained field-workers supplied by Dr. Davenport. In one instance one of these workers obtained information in regard to 3,300 members of a family group which had intermarried to such an extent that only five distinct families were represented. Of this number 76 were insane, 36 of them inmates in State hospitals. The following is a list of the various other abnormal individuals in this family:

Sexual offenders	50
Epileptics	5
Alcoholics	46
Feeble-minded	13
Cancer	19
Sarcoma	1
Blind	2
Congenital defective	1

"In practically every case investigated it is possible to obtain some information in not less than 200 members of a family, and sometimes a great many more. The field-workers have found no difficulty in obtain-

---

(2) Zeitschr. f. d. gesammte Neurol. und Psych., Vol. VII.

ing this information, and without exception, they have received courteous treatment from the individuals whom they have visited. We find that the families are much interested in the work and will give all the information possible. The field-worker becomes acquainted with the patient and usually talks with the patient before going to the family, and in this way carries messages back and forth and establishes friendly relations. They spend on an average fifteen days a month in the field. The rest of the time is spent at the hospital, writing up histories and making out charts. They do not attempt to make a diagnosis, but take down all facts given by the relatives. Wherever the family physicians know anything about the families, these are visited and their opinions also noted. Where relatives have been in the hospital reference is made to this, and a diagnosis made from the records when possible. When relatives have been in other hospitals, either in this state or in any other state, we have endeavored to obtain a copy of the records from these institutions.

"The patients in the hospital are catalogued according to communities, towns, cities, etc. When the field-worker goes to a certain district she has the names of the discharged patients who are living in that community. A visit is made to these discharged patients, and an endeavor made to learn something as to their condition. Often they find the environment such that it is necessary to report conditions to the hospital, and then advice can be given the family as to the right method to pursue to prevent a recurrence of the attack. This "after-care" work is a very important part of our field work and has resulted in much good to discharged patients. Several times during the year the field-workers devote all their time to looking up discharged patients. Besides looking up the heredity in families, they inquire into the habits, domestic relations, occupation and any other factors which are wanted by the physicians. In certain cases where the statements of the family were questioned, the field-worker went into the community and was able to prove or disprove these statements. We have now collected a large number of pedigrees, averaging 200 or more to a family."

Dr. Cotton gives a summary of the findings in five family charts collected and the following will serve as an example of the startling facts laid bare by this strictly objective and rational method.

Chart III (J. K.) is a summary which represents 200 individuals, fifteen of whom are insane, ten were in the Trenton State Hospital, twelve tuberculous, three neurotic, one feeble-minded, seventeen died in infancy, nineteen were alcoholic. Of the psychoses, we have six manic-depressive, four dementia precox, one questionable dementia precox, one senile paranoid condition, one imbecile, one feeble-minded, one unclassified. The paternal line is fairly good, with the exception of alcoholism. The maternal line, on the other hand, is very much affected. The mother is neurotic. One sister was a border-line case. Two sisters were manic-depressive cases. The mother had nine living children, three died in infancy, making a family of twelve. The mother had manic-depressive insanity from which she recovered and is now living at the age of 83. The father was alcoholic, had a sister who was in a senile paranoid condition and a brother with dementia precox. This brother had two children, both of whom are cases of dementia precox and inmates of this hospital. He married a woman put down as peculiar. The maternal cousin is a case of dementia precox in this hospital. As we see here, in the preceding generation the number of children exceed the large number of children of the present generation. In this family, out of five individuals who were insane in the grandparents and great-grandparents, only two were in institutions, while all the cases that were insane in the parents' children were committed to institutions, and this fact will be found to run through all our charts. One can conclude that in the preceding generations the percentage of the cases who were insane and were committed to an institution was much smaller than the percentage of the same class to-day. This has an important bearing on the apparent increase in the number in insane institutions at present, for I think it can be definitely shown that a large proportion of those who were insane in the community in previous generations were kept at home."

Unilateral "Thought Echo," a Contribution to the Study of Hallucinations, is the title of a paper by the prominent Vienna psychiatrist, Stransky.[3] A man 39 years old was admitted in 1899 on account of excitement, sleeplessness and threatening conduct following excessive drinking. He was exalted and flighty. He soon recovered and was discharged. He returned in January, 1911, and had again been intemperate. He had been hard of hearing in the right ear for years and stated that for five or six years there had been a blowing noise in the right ear, at times coupled with the sound of scolding voices but particularly he would hear his own thoughts repeated. He would hear this only in the right ear, and realized that it was an hallucination. He had no other hallucinations and no delusions of persecution. Otologic examination revealed middle-ear deafness in the right ear while the vestibular apparatus was normal. He heard the voices only three or four times a year, for two or three days at a time, while the blowing noise in the ear was constant. The patient denied that the hallucinatory periods had any relation to his sprees.

In connection with this case Stransky discusses the present theories regarding hallucinations and states that the majority of observers favor the "central" theory while others believe that hallucinations may originate in peripheral irritations. However, even the latter group of observers admit that the essential condition which permits hallucinations to occur must reside in the cortex. In the case just related alcoholism undoubtedly had been responsible for the disposition to hallucinations. The author believes that peripheral irritations, especially diseases of the ear, are important determining factors in the production of hallucinations.

DEMENTIA PRECOX.

Bodily Disturbances in Dementia Precox. Since serious discussion has arisen as to whether this disease is to be looked upon as a functional disturbance produced by mental causes or as an organic brain dis-

---

(3) Neurolog. Centralbl., Nov. 1, 1912.

ease the question whether symptoms exist which demand an organic explanation is of great importance. E. Meyer[4] reports some cases in point. A patient 29 years old whose brother also had dementia precox was first admitted to the Psychiatric Hospital in Königsberg in 1900, and then showed great motor restlessness. He apparently recovered but was readmitted in 1903 in an excited, incoherent state. He again seemed well but returned in 1911, talking in an incoherent monotonous manner and showing stereotype actions and grimaces. He had confused religious delusions. The pulse-rate then was between 70 and 80, but a few months later the pulse-rate for a month remained around 48. In February, 1912, the entire musculature became rigid with clonic jerkings, and the patient was cyanotic. He was conscious and resistive. The pulse-rate then was 80. Two days later the pulse-rate was 140. In the next few weeks he was in an apathetic and negativistic state, with a pulse-rate between 40 and 60. On getting up from the recumbent position the pulse-rate would be increased by 40 beats while in a few minutes after lying down it would decrease by 20 or 30 beats.

Meyer considers the case a certain one of dementia precox and points out that similar behavior of the pulse has been reported by others. Bradycardia, however, is rare as compared with the opposite condition, as a pulse-rate of 120 to 140 is not unusual. In such cases a variation of 20 or 30 beats on changing position is also common. Such lability of the pulse is commonly observed in functional neuroses, such as hysteria and neurathenia, and the author formerly looked upon it as a functional symptom belonging among the hysteriform manifestations of dementia precox.

It is more difficult to account for the bradycardia. The author considers the possibility of the brain swelling described by Reichardt but other signs of increased brain pressure were lacking in his case. The attack of muscular rigidity must be looked upon as a katatonic attack, but it was associated with slight fever and resembled the attacks occurring in paretic dementia. The

---

(4) Neurolog. Centralbl., Apr, 16, 1912,

lability of the pulse together with the frequent dermographia and cyanosis of the extremities may be looked upon as disturbances of the vasomotor apparatus and therefore not constituting organic symptoms. So-called "ovarian" neuralgia and mastodynia are also frequent and on the whole "functional" symptoms predominate.

The Eye Syndrome of Dementia Precox. H. H. Tyson and L. Pierce Clark,[5] ophthalmologist and neurologist respectively, studied the eyes in 115 consecutive cases, 109 of which were examined with the ophthalmoscope. Of these 55 were males and 54 females, ranging in age from 12 to 47 years. The fundus changes met with were divided in three classes:

1. Congestion of the discs; hyperemia and edema; dilated, dark-colored veins; slightly contracted arteries and blurring of the edges of the discs, all varying in degree. These charges constitute a low grade of perineuritis of the optic nerve. They were present in the right eye 62 times and in the left 67 times.
2. Congestion of the nasal side, with temporal pallor of discs, dilated veins, contracted arteries. These were seen in the right eye 10 times, and in the left eye 11 times.
3. Pallor of the discs, dilated veins, contracted arteries. These changes constitute anemia and partial atrophy of the optic nerve, and were found in the right eye in 37 cases, and in the left eye in 31 cases. The more marked changes were found in the more rapidly deteriorating types.

PARETIC DEMENTIA.

Paretic Dementia and Trauma. Supporting himself on personal experience with the enormous material of the London County Asylums, F. W. Mott[6] expresses the view that trauma plays an unimportant rôle, even as a contributing cause, in this disease.

"As a general result of my investigations I have come to the conclusion that head injury as an exciting cause was not of much importance either in causing

---

(5) Archiv. Ophthalmol., May, 1912.
(6) Brit. Med. Jour., Sept. 30, 1911.

the onset of general paralysis or in accelerating the symptoms. At the same time I have met with a few cases where undoubtedly head injury may have been the determining cause of the onset of the symptoms, and it is not unreasonable to suppose that a syphilitic, if he or she had not received some exciting cause, such as a blow on the head, might never have developed general paralysis, but these cases are rare.

"General paralysis may not be discovered until an accident or a head injury takes the patient to a hospital or infirmary. Thus I have known the case of a prostitute who received a blow in a drunken brawl. She was already a paralytic at large, but not until she was taken to the hospital or infirmary on account of the injury was the nature of her affection recognized; she was then sent to the asylum.

"My figures do not agree with those of Gudden, as there is practically no difference in the average age of death of those cases with a history of trauma and those without. There is one essential cause of general paralysis, and that is syphilis. All conditions which lead in a non-syphilitic subject to neurasthenia may act as exciting and contributory factors; they are sexual excesses, head injuries, alcoholism, speculating, gambling, business worries, leading to prolonged mental excitement and insomnia, in fact all forms of nervous excitement, singly or combined, which will lead to an exhausted nervous system, especially when acting upon an inborn neurotic and erotic temperament in the subject of acquired or inherited syphilis."

The Medico-Legal Relationships of Paretic Dementia formed the subject of a lecture by Sir George H. Savage.[8] He took up the subjects of brutal assaults, sexual offences, theft and other criminal acts. His discussion of the rôle of injury in the etiology and the resulting medico-legal complications is of interest.

"Recently a very important practical point has arisen in relationship to the part that injury may play in the production of general paralysis. In questions of compensation for injuries leading to death and also in cases of life insurance it may be contended that in-

---

(8) Lancet, Feb. 3, 1912.

jury alone would not have caused the disease. You will recognize that now the great majority of authorities look upon general paralysis and locomotor ataxia as almost certainly associated with, if not fully resulting from, syphilis. Yet I find even such an authority as Dr. F. W. Mott hesitating when asked if it is impossible for general paralysis to arise from accident alone. He, I think, agrees with my experience, which is, that in a very large majority of cases of general paralysis syphilis of long standing can be established, but that this disease alone is not the only factor. The question of heredity comes in, and the illuminating work of Mott inclines one to think that just as with alcoholic poisoning certain persons suffer in their brains while others do so in their general viscera, so with syphilis in some the stress of the disease falls upon the nerve centers, while in others it selects other parts such as the skin or the bones.

"To return to the main point, though syphilis is most commonly associated with general paralysis, yet it requires other elements to start the disease. I believe that meat diet and alcohol are factors in many cases, and incidentally I may say that one superintendent of a large asylum told me that with general paralytics he hardly ever gave a meat diet, and as a result the acute phases and the epileptiform seizures had vanished from the asylum.

"I have been in the habit of saying that syphilis seemed to ripen the brain for decay, and that any accident from within or from without might lead to the rottenness or decay. It therefore comes to this:—A man, who many years before had syphilis, was properly treated, and has been a healthy man, receives a blow on his head, which renders him insensible. He may recover from this, but within a few months some alteration is noticed in his conduct. If in one of the services, he gets into trouble by faults of irregularity or unpunctuality. He is irritable and forgetful; slowly the other signs of general paralysis appear and he is invalided. A claim is now made for disability depending on accident. Without the previous syphilis he would not have developed general paralysis, but without the accident he might never have developed it, or, at any rate, not for years. In my opinion, the man ought to be treated as if the accident were the efficient cause of disablement. In the same way in cases in which it is a question of life insurance, I presume a man who has had syphilis would have some addition made to his premium, but if he is following a life which is more than usually associated with risk of even slight accidents, such as being in the army or navy, I think a good deal ought to be added. I do not know of cases which have actually arisen in relationship to these points, but I know that they have arisen in cases in which pension questions had to be considered.''

Finally he takes up the question of testamentary capacity. As a general rule one is justified in saying that it is improbable that a paretic dement will make a good will, but he calls attention to the fact that English courts have supported wills that have been made by patients confined in asylums. The law demands that there must be sufficient memory to recognize the property concerned and its relationships, also normal memory of blood relations, freedom from delusions affecting either the property or the relations, and sufficient will power to prevent undue influence.

''The dangers in general paralysis are first on the emotional and unstable side, so that the so-called facility comes in, the last impression being followed. The patient will promise the last person whom he met all he possesses and more for no real consideration. The amorous tendency, too, easily leads to the giving largely to unworthy persons. The reckless extravagance depending on delusions of grandeur are very potent influences. The defective memory must also be considered. Here, then, we have commonly a person with defective memory, want of normal recognition of his relations and of his property, and therefore likely to go wrong. He is also very easily influenced. On the first aspect, then, of such cases one is justified in saying it is improbable that a general paralytic will make a good will, but even with all these symptoms he may almost automatically carry out in his will the feelings and judgments of his sane life. Therefore in each case the contents of the will will be found to influence a jury more than your opinion.''

It is also of great importance that a paretic dement may have remissions in which he is sufficiently well to make a legal will so it must not be concluded that the person in whom a diagnosis of paretic dementia has been made may not subsequently be able to make a will which will stand in court. In fact the author relates such a case.

OTHER PSYCHOSES.

Presenile Melancholia—A Psychologic and Clinical Study is the title of a monograph by Gaussen [9] based upon 100 cases, mostly from the clinic of Régis at Bordeaux. He takes up all phases of the subject in detail and formulates his chief conclusions as follows:

1. Melancholia is the most frequent psychosis of the critical age.
2. As in all psychoses heredity plays a rôle, yet we can plainly discern the effect of the critical age and all of the pathologic modifications connected with it.
3. In an ill-developed case melancholia may easily be confounded with neurasthenia.
4. When once developed, fear is the prominent symptom, often without delusions, but often with morbid obsessions and negativistic ideas connected with disturbances of kinesthesia, consciousness and the conception of the ego.
5. Presenile melancholia often ends in recovery; 68 of the author's 100 cases recovered. Nevertheless the prognosis of melancholia is grave both because of the possibility of the depression becoming chronic and of the danger of permanent mental defect, *i. e.,* dementia. The latter tendency is not due to the melancholia in itself, but to the arteriosclerotic process which frequently accompanies it. Therefore, treatment must be directed not only to the melancholia but to the phenomena of the menopause itself, the arteriosclerosis and the disturbances of the internal secretions.

---

(9) Thése de Bordeaux, 1911.

6. The theory of manic-depressive insanity is to a large extent based upon the frequency of recurrences in mania and melancholia. The theory further assumes that attacks of melancholia in youth and later in life have a tendency to recur. From this point of view, it is of interest to determine the frequency of previous attacks in cases of presenile melancholia between the ages of 45 and 65 years. Of the author's 100 cases, 90 positively had their first attack. If we, therefore, follow Dreyfus and Kraepelin in classing presenile melancholia under manic-depressive insanity, we must modify the statement that recurrences regularly occur in the latter disease. Other authors recognize an independent, isolated melancholia aside from manic-depressive insanity.

Folie à Deux. William A. Boyd[1] defines this as a mental disorder occurring in two or more predisposed individuals who have been intimately associated with each other. It is characterized by delusional ideas, particularly of a persecutory type, which may be transferred from one individual to another. Three types have been described, namely: 1. Imposed insanity; 2. simultaneous insanity; 3. communicated insanity.

1. The so-called imposed insanity was first described by Lasègue and Falret in 1877. They stated that delusional ideas of one person might be transferred to a sane individual, but it was necessary that the two should have been intimately associated and that the delusions should present some degree of probability in order that they might be accepted. They also noticed that the psychosis in the second individual did not run a typical course but tended to disappear as soon as the two were separated.

2. Simultaneous insanity. Examples are the famous epidemics of insanity in the middle ages and the smaller ones now occasionally produced by revivalists.

3. Communicated insanity. In this form the second individual accepts the delusional ideas of the first only after prolonged resistance, and the psychosis persists in the second person even after the two have been

---

(1) Med. Record, July 13, 1912.

separated. It is not considered absolutely essential that both cases should present the same psychosis.

Tuke classified the disorder as follows: 1. Cases in which an insane patient distinctly infects another person with the same mental disorder. 2. Cases in which a person becomes insane from companionship, not in consequence of direct transference of morbid ideas, but in consequence of the shock arising out of the painful impressions caused by witnessing the attack, or by the strain of nursing a patient. 3. Cases in which two or more persons become insane simultaneously from the same cause. 4. Cases in which one lunatic infects another lunatic with his or her special delusion.

At the conclusion of an article on *folie à deux,* among other conclusions, Tuke arrived at the following: 1. In the majority of cases, those who become insane in consequence of association with the insane are neurotic or somewhat feeble-minded. 2. Females are more commonly affected than males. 3. The most common form which cases of communicated mental disorder assume is that of delusion, or of being entitled to property of which they are defrauded by their enemies. 4. A young person is more likely to adopt the delusions of an older person than *vice versa,* especially if the latter be a relative. 5. It is often difficult to determine to what extent the person who is the second to become insane affects in his turn the mental condition of the primary agent.

Boyd reports the case of a mother and daughter which was unusual in that the younger person, the daughter, seemed to be the active agent or "parasite." The daughter was 24 years old, had spent most of her childhood in assisting her mother with the household work. At about 20 she began to act strangely, seemed to be suspicious, and had ideas of "reference"; that is she thought people on street-cars, at Sunday school and at the office where she worked as a stenographer, laughed at her and made slighting remarks about her. She developed auditory hallucinations and feared that she would be arrested, taken away, locked up and killed.

Gradually the mother developed similar symptoms. She would not eat, feared that something was going to happen—that she was going to be punished. She believed that her daughter had a "spell" over her which she could not break and wanted the daughter sent away for fear that this influence might make her mad. She was self-willed, and would threaten to kill herself if her husband did not allow her to do as she pleased. She would often mutter to herself in a low tone of voice, and there was some retardation in speech and movement, and her emotional field was very unstable. Marked ideas of reference were present; she thought people were trying to injure herself and her daughter. She was suspicious of the conversation and actions of every one except her daughter. Auditory hallucinations were frequent. She heard voices under the window talking to her and about her. She also heard voices of people in the shrubbery about the house, who were eavesdropping, and who she thought were planning to rob the house. Mother and daughter spent much of the time reading the Bible together; this was unusual. They would sit beside each other all day and often until after midnight, talking to each other of their imaginary troubles in whispers, fearing that they might be overheard. They believed that everyone was talking about them. The mother said that the neighbors called her daughter a bad character and that she would have to share the disgrace. They were superstitious, wanting the dog and cat killed and their blood sprinkled upon the doorsteps. They both firmly believed that their own presence about the house brought bad luck. They expressed the delusion that the farm was going to be taken away from them, and one very rainy day, without protection of umbrella or raincoat, they started to the house of their pastor to ask his intercession in this matter. Each patient seemed to wield a great influence over the other; the mother obeyed her daughter implicitly; the daughter in turn would not obey her father unless she first asked her mother's consent. By the fifth week after the acute onset, neither of the patients would do any housework, and the husband found it necessary to prepare his own meals."

Both were admitted to the insane hospital. After

5 months the mother was taken home and a few weeks later was reported to be practically well. The daughter, on the other hand, did not improve and was considered a case of the hebephrenic form of dementia precox.

Transitory Mental Confusion and Delirium in Old Age occasionally occur and this should teach us that the occurrence of delirium or confusion in an old person does not always mean incurable and hopeless senile insanity. Charles W. Burr[2] reports a number of such cases.

Case 1. A man 70 years old became excited and incoherent while at lunch at his club. He delivered a very dramatic but incoherent address to an imaginary audience and did not recognize members of his family. He was given a hypodermic of morphine and bled a pint from the arm. The next morning his mind was clear but part of the previous day was a blank to him.

"It is a good rule always to bleed a plethoric man who has any sudden cerebral attack, with flushed face, mental excitement or coma, hard swollen temporal arteries and a bounding pulse whether there is palsy or not and whether there are convulsions or not. It is also a good rule to give such a patient at the time of bleeding, if there is mental excitement, not of course if coma be present, a dose of morphine hypodermatically, even though the kidneys are not doing their proper work. It is often impossible at the time to tell whether a stroke is due to cerebral hemorrhage or is uremic, that is, due to an auto-intoxication dependent on kidney disease without a gross cerebral lesion. Uremia may produce a condition indistinguishable from cerebral hemorrhage. Bleeding can do no harm in the latter case and in the former often does much good.

"Offhand one would scarcely think it possible to mistake aphasia for delirium, but transitory attacks, either total or causing jargon-speech, may cause such an appearance of mental confusion as to mislead the observer entirely. This is shown in Case 2.

Case 2. A woman, aged 86, well educated, and with rather more than the average of intelligence but very

---

(2) Jour. Amer. Med. Assoc., Dec. 30, 1911.

spare in build and with no more blood than she needed, was suddenly seized—while seemingly in excellent health and without any sudden external stress—with total speechlessness lasting for a few minutes and followed by jargon-speech. She became quite excited, talked gibberish, and for about an hour made wild gesticulations which no one could understand. There was no period of unconsciousness, no palsy except possibly a slight weakness of the movements of the right side of the face, and no convulsions or spasm. The patient appeared to understand nothing said to her. At the end of an hour speech returned, not entirely but enough for her to make herself clearly understood. She rapidly became mentally and emotionally quiet, and said she had heard and understood all that was said to her; indeed she told me much of it and related several things that had happened in the room, but, as she explained, her inability to make herself understood by speech so frightened her that she could not control herself and make herself show by gestures and by obeying commands that she understood what was said to her. Her real trouble was a temporary aphasia due either to a local intoxication or, and for many reasons this seems more probable, a partial thrombosis.

Since the first attack, she has had a thrombus which caused a temporary right-sided hemiplegia, and later another which has left her with a right-sided hemiplegia and partial motor aphasia. Neither of the last two attacks was accompanied by any mental excitement and now—two years after the first attack—her mind is clear and judgment good, though palsy and some motor aphasia continue.

The Burden of Feeble-Mindedness is the title of an address delivered by Walter E. Fernald[3] before the *Massachusetts Medical Society.* He estimates there are two feeble-minded persons to every 1,000 of the population. As to causes he says:

"The various known causes of feeble-mindedness occur in two main groups—the hereditary and the accidental. The hereditary cases are those where the person is feeble-minded because his parents or other ancestors

---

(3) Boston Med. and Surg. Jour., June 20, 1912.

were feeble-minded. The accidental group includes those who are feeble-minded as a result of environmental causes, without hereditary influence.

"The hereditary cases are the most numerous. The recent intensive study of the family histories of large numbers of the feeble-minded by Goddard, Davenport and Tredgold show that, in at least 80 per cent. of these cases, the mental defect had been preceded by other cases of defect in the immediate family line. Goddard finds that 65 per cent. of his institution cases had one or both parents actually feeble-minded. It is believed that this hereditary defect is the result of protoplasmic defect in the germ-plasm of the family stock.

"There is no doubt as to the potency and certainty of this hereditary tendency. Often the feeble-minded child represents a feeble-minded family. Davenport believes that aside from the Mongolian type, probably no imbecile is born except of parents who, if not mentally defective themselves, both carry mental defect in their germ-plasm.

"Among the probable accidental or environmental causes of feeble-mindedness are injuries to the head at birth, blows or falls in infancy, inflammatory brain disease, toxemia from infectious diseases, abnormal mental or physical conditions of the parents, etc., or the absence of certain vital substances from the blood, as in cretinism. Cases of feeble-mindedness often occur in families where there has been no mental disease or defect for several generations, but even where the exciting cause is undoubtedly accidental, there is often a strong hereditary predisposition. Similar injuries or causes in sound families do not result in feeble-mindedness. In the majority of these cases the environmental causes are only accessory. The real origin of the disease lies in the defect of the germ-plasm.

"Certain types of defect are usually if not always due to accidental or sporadic causes, *viz.*, the Mongolian, hydrocephalic, post-meningitic, the cerebral hemorrhagic, etc. Acquired characteristics are not likely to be transmitted, but there is reason for the belief that alcoholism, syphilis, tuberculosis and other environmental factors may initiate germinal variation which may become hereditary. The cases of purely accidental origin with no morbid heredity are not likely to be followed by other cases in that family. The purely accidental cases themselves would probably beget normal progeny.''

As to measures for reducing the number of feebleminded in the future Fernald says:

''It is probable that intelligent study of the whole problem on a large scale will furnish data for adequate treatment and control. The full problem should be stated by a complete and permanent census of the feeble-minded of the entire state. This is possible by the co-operation of physicians, teachers, social workers, court and prison officials, local authorities, etc. Such registration would be analogous to the required notification and registration of contagious and infectious diseases, and would be the first step in the regulation and elimination of defective strains from the community. The State Board of Insanity has already begun an informal and tentative census of this sort.

''In the light of our present knowledge, the only way to reduce the number of the feeble-minded is to prevent their birth. The perpetuation of defective family stocks should be inhibited. This would be possible to a great extent if every feeble-minded person and every potential 'carrier' of the defective germ-plasm could be prevented from parenthood.

''There is already a strong popular demand for the logical and thorough application of our present knowledge of the laws of morbid heredity in the way of prevention. This state has begun the policy of the segregation of the feeble-minded, especially those of child-bearing age. This segregation carried out thoroughly for a generation would largely reduce the amount of feeble-mindedness. The high-grade female imbecile group is the most dangerous class. They are not capable of becoming desirable or safe members of the community. They are never able to support themselves. They are certain to become sexual offenders and to spread venereal disease or to give birth to degenerate children. Their numerous progeny usually become public charges as diseased or neglected children, imbeciles, epileptics, juvenile delinquents or later on as adult paupers or criminals. The segregation of this class should be rapidly extended until all not adequately guarded at home are placed under strict sexual quarantine. Hundreds of known cases of this sort are now at large because the institutions are overcrowded.''

On the interesting subject of sterilization he has this to say: ''Compulsory surgical sterilization of all defectives is proposed as a radical method for preventing the hereditary transmission of feeble-mindedness. At least six states have passed laws authorizing or requiring this operation. In no state, however, has this remedy been applied on a large scale. There are many objections to this plan. The friends of the patients are not willing to have the operation performed. The normal 'carriers' of defect would not be affected. The presence of these sterile people in the community, with unimpaired sexual desire and capacity, would be a direct encouragement of vice and a prolific source of venereal disease. Sterilization would not be a safe and effective substitute for permanent segregation and control.

''It is probable that education in the broadest sense will be the most effective method in a rational movement for the diminution of feeble-mindedness. The public generally should be intelligently informed as to the extent, causation and significance by means of suitable literature, popular lectures and other means. There is now great demand for such information from women's clubs, church societies, charitable organizations, etc. General knowledge of this subject in a community will insure the rational protection and control of the feeble-minded persons in that community.''

INDEX.

Acid, diacetic, in cerebrospinal fluid, 7.
Albumins and xanthochroma, increase of, in spinal fluid, 8.
Alcoholism, chronic, pupillary disturbances in, 156.
Amyotonia congenita, is it a sequel of poliomyelitis during fetal life, 178.
  congenita, metabolic observations on, 179.
Anal eroticism, 16.
Ankle reflexes and larynx, condition of, in paralysis agitans, 196.
Application of psycho-analysis to insanity, 14.
Athetosis and motor disturbances, resection of motor nerves in, 58.
Atrophy, optic, of tabes, visual fields in, 136.
  primary optic, blindness due to, 107.
  progressive muscular, from overwork, 141.
Avulsion of anterior and posterior spinal roots for brachial plexus paralysis, 159.

Beri-beri, 167.
  experiments on causation of, 171.
Blindness due to primary optic atrophy, 107.
  serous meningitis and brain disease as cause of, 38.
Body, hemihypertrophy of, 187, 189, 192.
Brain and cord, cysticercus of, and cysticercus meningitis, 40.
  and meninges, diseases of, 38.
  disease and serous meningitis as cause of blindness, 38.
  histologic examination of, during life by means of brain puncture, 52.
  lesions produced by electricity, 202.
  multiple hemangio-perithelioma of, 77.
  puncture, histologic examination of brain during life by means of, 52.
  syphilis, is trephining indicated in, 73.
  tumor and hydrocephalus without enlargement of head, differential diagnosis, by x-rays, 74.
  tumor, history of, 107.
  tumor, value of tuning-fork tests in early diagnosis of, 75.
Bromid versus hygienic treatment of epilepsy, 20.

Cerebral lesions, sensory disturbances from, 43.
  localization, 43.
  syphilis and trauma, 59.
Cerebrospinal fluid, chemical tests in, 8.
  fluid, diacetic acid in, 7.
Character, defects of, value of psycho-analysis in correction of, 19.

Childhood, narcoleptic attacks of, resembling epilepsy, 32.
Cholesterin crystals in spinal fluid, 6.
Chordoma, malignant, 78.
Chorea, mental symptoms of, 33.
  minor, tendon reflexes in, 34.
Cirrhosis, liver, a familiar nervous disease associated with, 91.
Claudication, intermittent, lumbar type of, 146.
  intermittent, of spinal cord, 145.
Cog-wheel resistance of extremities in paralysis agitans, 195.
Color hearing, 10.
Confusion, transitory mental, and delirium in old age, 224.
Cord and brain, cysticercus of, and cysticercus meningitis, 40.
Crotalin treatment of epilepsy, 23.
Cysticercus meningitis and cysticercus of brain and cord, 40.
  of brain and cord and cysticercus meningitis, 40.
Cytology of spinal fluid, 6.

Decompression of spinal cord, 147.
Defects of character, value of psycho-analysis in correction of, 19.
Degeneration, progressive lenticular, 91.
Delirium and confusion in old age, 224.
Dementia, paretic, and trauma, 216.
  paretic, medicolegal relationships of, 217.
  paretic, salvarsan in, 69.
  precox, bodily disturbances in, 214.
  precox, eye syndrome of, 216.
Dermato-myositis, a case of, 177.
Diacetic acid in cerebrospinal fluid, 7.
Diagnosis, differential, between hydrocephalus without enlargement of head and brain tumor by x-rays, 74.
  of brain tumor by tuning-fork tests, 75.
Disease, pituitary, infantilism in, 107.
Diseases of brain and meninges, 38.
  of muscles, 177.
  of the nervous system, symptomatology of, 5.
  of peripheral nerves, 152.
  of spinal cord, 111.
Dust as a carrier of poliomyelitis infection, 113.
Dyspituitarism, classification of states of, 104.

Edema, angioneurotic, fatal, with necropsy, 193.
Electricity, brain lesions produced by, 202.
Epilepsy, crotalin treatment of, 23.
  hygienic versus bromid treatment of, 20.
  narcoleptic attacks in childhood resembling, 32.
  skepticism as to surgical treatment of, 30.
  surgical treatment of, 25.
Eroticism, anal, 16.
Etiology of so-called rheumatic facial paralysis, 152.
Evolution of Freud's psychoanalytic method, 10.
Examination for spirocheta pallida, 61.
Eye syndrome of dementia precox, 216.

Face, loss of emotional movement of, with preservation or slight involvement of voluntary movement in partial paralysis of facial nerve, 155.

Facial hemiatrophy, 190.
Feeble-mindedness, burden of, 225.
Flies in transmission of poliomyelitis, rôle of, 114.
Folie à deux, 221.
Fossa, posterior cranial, tumors of, disturbances in, 52.
Freud's psycho-analytic method and its evolution, 10.

Ganglia, basal, 91.
Ganglioma embryonale sympathicum, 176.
Globulin in spinal fluid, test for, 60.
Gold chlorid, test with spinal fluid in syphilitic nervous diseases by, 61.

Hallucination of smell, 52.
Hallucinations, unilateral thought echo a contribution to study of, 214.
Head, value of decompressive operation on, 86.
Hearing, color, 10.
Hemangio-perithelioma, multiple, of brain, 77.
Hemiatrophy, facial, 190.
  from scleroderma, 191.
Hemihypertrophies and hemiatrophies, 187.
Hemihypertrophy of body, 187, 189, 192.
Hemisphere, dominating right, in right-handed person, 53.
Heredity and mental diseases, value of field work in study of, 210.
Hexamethylenamin in poliomyelitis, value of, 133.
Hydrocephalus without enlargement of head and brain tumor, differential diagnosis between, by x-rays, 74.
Hypophysis, 101.
Hypopituitarism, histologic studies in, 102.

Infantilism in pituitary disease, 107.
Infectious diseases, neuritis due to, 164.
Insanity, application of psychoanalysis to, 14.
Iowa epidemics of poliomyelitis of 1910 and 1911, 132.

Laminectomy with negative findings of suspected tumor of eighth thoracic segment, 150.
  with negative findings of suspected tumor of spinal cord, 151.
Larynx and ankle reflexes, condition of, in paralysis agitans, 196.
Lenticular degeneration, progressive, 91.
Lesions, cerebral, sensory disturbances from, 43.
  visceral, in human cases of poliomyelitis, 116.
Liver cirrhosis, a familiar nervous disease associated with, 91.

Medicolegal relationships of paretic dementia, 217.
Melancholia, presenile, 220.
Meninges and brain, diseases of, 38.
Meningitis, cysticercus, and cysticercus of brain and cord, 40.
  serous, and other temporary organic brain diseases as cause of blindness, 38.
Mental symptoms of chorea, 33.
Metadromal progression, 198.
Migraine, ophthalmic, unilateral rigidity of pupil in, 35.
Monkeys, clinical picture of experimental poliomyelitis in, 128.
Muscles, diseases of, 177.
Myotonia congenita, or Thomsen's disease, 180.

Narcoleptic attacks in childhood resembling epilepsy, 32.

Necropsy, fatal angionéurotic edema with, 193.
Nerve recurrences after salvarsan, 69, 71.
  direct trauma to, neuritis dependent on, 162.
  intercostal, avulsion of, for gastric crises, 137.
  motor, partial resection of, in athetosis and other motor disturbances, 58.
  peripheral, diseases of, 152.
Nervous diseases of tobacco smokers, 182.
  system, non-syphilitic tumors of, positive Wassermann reaction in, 77.
  system, syphilitic diseases of, salvarsan in treatment of, 64.
Neuritis dependent on direct trauma to the nerves, 162.
  dependent on exposure to poisonous substances, 162.
  dependent on fatigue of muscles, 163.
  due to alcoholic and infectious diseases, 164.
  multiplex cutanea, 173.
  occupation, 162.
  syphilitic multiple, does it exist, 165.
Neurofibromatosis, 174.
  cause of pyloric stenosis, 175.
  sudden eruption of, 175.
Neuroses, functional, training-camp method in treatment of, 36.
  psycho-analysis in, 10.
Nicotine, toxicology of, 186.
Nonne-Apelt ''Phase 1'' test for globulin in spinal fluid, 60.
Norway, poliomyelitis in, 130.

Occupation neuritis, 162.
Operation, decompressive, value of, on head, 86.
  Franke's, for gastric crises by avulsion of intercostal nerves, 137.
Oppenheim's experience with salvarsan, 70.
Osteochondritis of twelfth dorsal vertebra, 150.

Paralysis agitans, a new diagnostic sign in, 195.
  agitans, condition of larynx and ankle reflexes in, 196.
  agitans, metadromal progression in, 198.
  agitans, resection of posterior roots, for relief of rigidity in, 197.
  agitans, Stellwag's sign in, 197.
  brachial plexus, with avulsion of anterior and posterior spinal roots, 159.
  infantile: See also Poliomyelitis.
  infantile, determining the possibility of transmission of, 126.
  infantile, presence of microbe of, in human beings, 124.
  peripheral facial, electric reactions in, 153.
  so-called rheumatic facial etiology of, 152.
  spastic cerebral, rhizotomy in treatment of, 55.
Paraplegia dolorosa of three years' duration, 150.
Pellagra, 202.
Person, right-handed, dominating right henisphere in, 53.
Pineal body, teratoma of, 109.
Pituitary body and its disorders, 101.
  disease, infantilism in, 107.
Poliomyelitis: See also Paralysis, infantile.
  acute anterior, mode of infection, 111.
  clinical study of, 119.
  epidemics of Iowa, 132.
  experimental, in monkeys, clinical picture of, 128.
  hexamethylenamin in, 133.
  histologic changes in, 116.
  histologic examination of mucous membranes in, 129.

infection, dust as a carrier of, 113.
in Norway, 130.
is amyotonia congenita a sequel of, during fetal life, 178.
possibility of transmission of by blood-sucking animals, 127.
rôle of flies in transmission of, 114.
sensory disturbances in, 117.
Swedish epidemic of, of 1911, 123.
visceral lesions in, 116.
Pseudo-sclerosis, new symptom-complex resembling, 98.
Psychiatry, value of psychology in, 205.
Psycho-analysis in the neuroses, 10.
application of, to insanity, 14.
value of, in correction of defects of character, 19.
Psychology in psychiatry, value of, 205.
teaching of, in medical schools, 206, 207.
Puncture, brain, histologic examination of brain during life by means of, 52.
Pupil disturbances in chronic alcoholism, 156.
unilateral rigidity of, in ophthalmic migraine, 35.

Rattle-snake venom in epilepsy, 23.
Reaction, positive Wassermann, in non-syphilitic tumors of nervous system, 77.
Reactions, electric, in peripheral facial paralysis, 153.
Reflex, new, the wrist sign, 5.
tendon, in chorea minor, 34.
Resection of posterior roots for relief of rigidity in paralysis agitans, 197.
partial, of motor nerves, in athetosis and motor disturbances, 58.
Rhizotomy in treatment of spastic cerebral paralysis, 55.
Rigidity, unilateral, of pupil, in ophthalmic migraine, 35.

Salvarsan in paretic dementia, 69.
in treatment of syphilitic diseases of nervous system, 64.
nerve recurrences after, 69, 71.
Oppenheim's experience with, 70.
therapeutic results with, 68.
Scleroderma, hemiatrophy from, 191.
Sclerosis, essential or primary lateral, 137.
multiple, clinical types of, 138.
Section of posterior nerve roots: See Rhizotomy, 55.
Sensory disturbances from cerebral lesions, 43.
disturbances in poliomyelitis, 117.
Skepticism as to surgical treatment of epilepsy, 30.
Smell, hallucination of, 52.
Spinal cord decompression, 147.
cord, diseases of, 111.
cord, intermittent claudication of, 145.
cord, traumatic diseases of, 141.
cord, tuberculosis of, 134.
fluid, cholesterin crystals in, 6.
fluid, cytology of, 6.
fluid, globulin in, test for, 60.
fluid, increase of albumins and xanthochroma of, 8.
fluid, test with, in syphilitic nervous diseases by gold chlorid, 61.
Spirocheta pallida, examination for, 61.
Stellwag's sign in paralysis agitans, 197.

Stenosis, pyloric, caused by neurofibromatosis, 175.
Sunstroke, nervous symptoms following, 198.
Surgical treatment of epilepsy, 25.
Swedish epidemic of poliomyelitis of 1911, 123.
Symptomatology of diseases of the nervous system, 5.
Symptom-complex, new, resembling pseudo-sclerosis, 98.
Syphilis, cerebral, and trauma, 59.
  of brain, is trephining indicated in, 73.

Tabes dorsalis, visual fields in optic atrophy of, 136.
Taste, disturbances of, in tumors of posterior cranial fossa, 52.
Tendon reflexes in chorea minor, 34.
Teratoma of pineal body, 109.
Test, Nonne-Apelt ''Phase 1,''
  for globulin in spinal fluid, 60.
  with spinal fluid in syphilitic nervous diseases by means of gold chlorid, 61.
Tests, chemical, of cerebrospinal fluid, 8.
  tuning-fork, value of, in early diagnosis of brain tumor, 75.
Tetany, signs of, 176.
Thomsen's disease or myotonia congenita, 180.
Thought echo, unilateral, a contribution to study of hallucinations, 214.
Tobacco smokers, nervous diseases of, 182.
Toxicology of nicotine, 186.
Training-camp method in treatment of functional neuroses, 36.
Transmission of poliomyelitis by flies, 114.
Trauma and paretic dementia, 216.
  cerebral syphilis and, 59.
Treatment, crotalin, of epilepsy, 23.
  hygienic versus bromid, in epilepsy, 20.
  of functional neuroses by training-camp method, 36.
  of poliomyelitis, 133.
  of spastic cerebral paralysis by rhizotomy, 55.
  of syphilitic diseases of nervous system by salvarsan, 61.
  surgical, of epilepsy, 25.
Trephining in brain syphilis, is it indicated, 73.
Tuberculosis of spinal cord, 134.
Tumor of brain and hydrocephalus without enlargement of head, differential diagnosis between, by x-rays, 74.
  of brain, value of tuning-fork tests in early diagnosis of, 75.
Tumors, non-syphilitic, of nervous system, positive Wasser mann reaction in, 77.
  of fourth ventricle, 79.
  of posterior cranial fossa, disturbances of taste in, 52.
  pineal, metabolic symptoms of, 110.

Ventricle, fourth, tumors of, 79.
Von Recklinghausen's disease, familial, 174.

Wassermann reaction, positive, in non-syphilitic tumors of central nervous system, 77.
Wrist sign, a new reflex, 5.

Xanthochroma and albumins, increase of, in spinal fluid, 8.
X-rays, differential diagnosis between hydrocephalus without enlargement of head and brain tumor by, 74.

INDEX TO AUTHORS.

Allen, 149.
Anderson, 115.
Assmann, 68.

Bailey, P., 109, 148.
Bartels, 7.
Bassi, 58.
Bassoe, P., 189.
Batten, F. E., 177.
Battiotini, 75.
Beltz, L., 60.
Benario, 71.
Bertolotti, M., 74.
Bierring, W. L., 132.
Bonhoeffer, K., 79.
Boyd, W. A., 221.
Bregman, L. E., 34.
Brill, A. A., 16.
Bulloch, 194.
Burr, C. W., 224.

Cagiati, 190.
Campbell, C. M., 14.
Chaddock, 5.
Clark, L. P., 20, 55, 216.
Cohn, T., 153.
Cooper, E. A., 171.
Cotton, H. A., 210.
Crowe, S. J., 133.
Cushing, 101.

Déjérine, 44, 145.
Demanche, R., 165.
Dochez, 119.
Doerr, C., 134.
Dössecker, 72.
Draper, 116, 119.
Dreyer, F., 59.
Dubourg, J., 175.

Edsall, 201.
Elsberg, C. A., 148.
Erb, 141, 143.

Fernald, W. E., 225.
Findlay, L., 180.
Fleischer, B., 98.
Flexner, 116.
Flexner, S., 111.
Foerster, O., 52.
v. Frankl-Hochwart, 182.
Franz, S. I., 207.
Frazier, C. H., 25, 30, 86.
Friedmann, M., 32.
Frölich, 186.
Frost, 115.
Funk, C., 171.

Gatz-Emanuel, E., 154.
Gaussen, 220.
Geist, 192.
Giese, 144.
Gittings, J. C., 179.
Glikin, 65.
Glynn, 61.
Goldstein, K., 40.
Graffner, 196.
Greenfield, J. G., 8.
Griffith, 180.
Grunberger, 7.

Harbitz, 130.
Harbitz, F., 193.
Head, H., 43.
Hewlett, A. W., 107.
Hoffmann, J., 165.
Holinger, J., 75.
Holmes, G., 43.
Holst, A., 167.
Horsley, V., 150.
Hunt, J. R., 147.

Jelliffe, 68.
Jelliffe, S. E., 109.
Jendrassik, 152.
Jolly, P., 33.

Josefson, 133.
Josefson, A., 126.

Ketly, K., 152.
Kling, C., 124, 127, 128, 129.
Knapp, P. C., 191.
Krause, P., 119.
Kutzinski, 52.

Lafora, G. R., 84.
Landsteiner, 127.
Lange, C., 61.
Langenbeck, K., 136.
Larkin, 78.
Leblanc, 137.
Leriche, 197.
Leriche, R., 175.
Levaditi, 127.
Lewandowsky, 54.
Ludlum, S. D. W., 137.

Macnaughton, N. S., 174.
Marburg, 110, 178.
Marinesco, G., 10.
Mattirollo, 75.
Medea, 58.
Menard, P. J., 165.
Mendel, K., 53.
Meyer, A., 205.
Meyer, E., 145, 215.
Mirowsky, M., 162.
Mohr, R., 6.
Mott, F. W., 216.
Moyer, H. N., 195.
Muskens, 38.

Neustaedter, M., 113.
Newmark, L., 77.
Nonne, 64, 156.

Oppenheim, 138.
Orbison, T. J., 36.
Ottendorff, 144.

Pastia, 127.
Peabody, 116, 119.
Pemberton, R., 179.
Peritz, 65.
Pettersson, A., 124, 127, 128, 129.
Phillips, 61.
Pick, L., 177.
Pierret, 175.
Prince, M., 209.
Putnam, J. J., 10.

Radash, 202.
Raven, W., 8.
Reichmann, 8.
Rolleston, J. D., 174.
Rosenau, M. J., 114.
Roussy, 44.
Rudin, 211.

Sabrués, J., 175.
Savage, G. H., 217.
Schlesinger, 134.
Schlesinger, H., 173, 176.
Scholz, B., 52.
Schultze, F., 197.
Schwarz, 7.
Scripture, E. W., 19.
Sergeant, 175.
Sicard, 137.
Skillern, P. G., 159.
Snellen, 38.
Spangler, R. H., 23, 24.
Spiller, 74, 150.
Spiller, W. G., 78, 155.
Spitzka, 202.
Steinert, 165.
Stier, 187, 190.
Stransky, 214.
Strassman, 61.
Strümpell, 117.

Taylor, A. S., 55.
Tilney, F., 198.
Tucker, B. R., 202.
Turner, J., 24.
Tyson, H. H., 216.

Ullmann, 72.

Van Gehuchten, 198.
Völsch, 101.

Walter, R., 116.
Watson, J. B., 207.
Weiler, 156.
Weisenburg, T. H., 198.
Wernstedt, W., 124, 127, 128, 129.
Westphal, 35.
Wilson, S. A. K., 91.

Zipperling, 59.